EXHIBIT 2

Expert Report of
Barbara D. Beck, Ph.D., DABT, ATS, ERT
in the Matter of
Hardwick *v.* 3M Company, *et al*.

Case No. 2:18-CV-1185

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Abbreviations

ADME	Absorption, Distribution, Metabolism, and Excretion
AFFF	Aqueous Film-Forming Foam
APFO	Ammonium Perfluorooctanoate
AR-AFFF	Alcohol-Resistant Aqueous Film-Forming Foam
AR-FFFP	Alcohol-Resistant Film-Forming Fluoroprotein Foam
ATSDR	Agency for Toxic Substances and Disease Registry
CSF	Cancer Slope Factor
diPAP	Polyfluoroalkyl Phosphoric Acid Diester
FEP	Fluorinated Ethylene Propylene
FFFP	Film-Forming Fluoroprotein Foam
FOSA	Perfluoroalkyl Sulfonamide
FP	Fluoroprotein Foam
FPAR	Alcohol-Resistant Fluoroprotein Foam
FTCA	Fluorotelomer Carboxylic Acid
FTOH	Fluorotelomer Alcohol
FTS	Fluorotelomer Sulfonate
FTUCA	Fluorotelomer Unsaturated Carboxylic Acid
GFR	Glomerular Filtration Rate
ITRC	Interstate Technology and Regulatory Council
IUR	Inhalation Unit Risk
LOAEL	Lowest Observed Adverse Effect Level
LOQ	Limit of Quantitation
monoPAP	Fluorotelomer Phosphate Monoester
N-EtFOSA	N-Ethyl Perfluorooctane Sulfonamide
NHANES	National Health and Nutrition Examination Survey
NOAEL	No Observed Adverse Effect Level
OAT	Organic Anion Transporter
PBPK	Physiologically Based Pharmacokinetic
PFA	Perfluoroalkoxy
PFAA	Perfluoroalkyl Acid
PFAS	Per- and Polyfluoroalkyl Substances
PFBA	Perfluorobutanoic Acid
PFBS	Perfluorobutane Sulfonate
PFBuS	Perfluorobutane Sulfonic Acid
PFCA	Perfluoroalkyl Carboxylic Acid
PFDA	Perfluorodecanoic Acid
PFECA	Perfluoroether Carboxylic Acid
PFHpA	Perfluoroheptanoic Acid
PFHS	Perfluorohexane Sulfonate
PFHxA	Perfluorohexanoic Acid
PFHxS	Perfluorohexane Sulfonate
PFNA	Perfluorononanoic Acid
PFO	Perfluorooctanoate

PFOA	Perfluorooctanoic Acid
PFOS	Perfluorooctane Sulfonate
PFOSA	Perfluorooctane Sulfonamide
PFPA	Perfluorophosphonic Acid
PFPiA	Perfluoroalkyl Phosphinic Acid
PFSA	Perfluoroalkane Sulfonic Acid
PFSiA	Perfluoroalkyl Sulfinic Acid
PFUnDA	Perfluoroundecanoic Acid
ppq	Parts Per Quadrillion
ppt	Parts Per Trillion
PTFE	Polytetrafluoroethylene
RfC	Reference Concentration
RfD	Reference Dose
RSL	Regional Screening Level
SAB	Science Advisory Board
UF	Uncertainty Factor
US EPA	United States Environmental Protection Agency
US FDA	US Food and Drug Administration
US	United States
WWTP	Wastewater Treatment Plant

1 Introduction

1.1 Case Overview

This matter, Hardwick *vs.* 3M Company, *et al.*, consists of claims that the Plaintiff, Mr. Kevin Hardwick, and others have been exposed to per- and polyfluoroalkyl substances (collectively, "PFAS")[1] from various Defendants who have marketed, developed, manufactured, distributed, released, sold, and/or used PFAS (Taft Stettinius & Hollister, LLP, 2019a). The Defendants in this matter are 3M Company; E. I. du Pont de Nemours and Company; The Chemours Company; Archroma Management, LLC; Arkema, Inc.; Arkema France, S.A.; AGC Chemicals Americas, Inc.; Daikin Industries Ltd.; Daikin America, Inc.; and Solvay Specialty Polymers, USA, LLC (Taft Stettinius & Hollister, LLP, 2019a).

It is my understanding that the Plaintiff is requesting certification of a nationwide class of any individuals residing in the United States (US) at the time of class certification for one year or more since 1977 and who have 0.05 parts per trillion (ppt) or more of perfluorooctanoic acid (PFOA) and 0.05 ppt or more of any other PFAS in their blood serum (Taft Stettinius & Hollister, LLP, 2019b). There are no health effect studies of human populations with serum levels of any PFAS as low as 0.05 ppt, which is the equivalent of 50 parts per quadrillion (ppq). The Plaintiff is a firefighter and alleges exposure to one or more PFAS from firefighting foams and materials used to coat firefighting equipment and gear (Taft Stettinius & Hollister, LLP, 2019a). The Plaintiff claims that exposures to PFAS from the Defendants have resulted in contamination of his blood with PFAS and biopersistence and bioaccumulation of PFAS within his body (Taft Stettinius & Hollister, LLP, 2019a). The Plaintiff seeks to have the Defendants pay for a program that involves studying the nationwide impacts, if any, of the thousands of different PFAS and medical monitoring of the proposed class (Taft Stettinius & Hollister, LLP, 2019b).

I was retained by the Defendants in this matter to evaluate, to a reasonable degree of scientific certainty, the extent to which the characteristics of the proposed class may vary from an exposure, dose, and risk perspective with respect to the many different PFAS that are the subject of the Plaintiff's claims.[2] My *curriculum vitae* is attached as Appendix A. Gradient is compensated for my time at the rate of $645/hour. To the extent that I receive additional materials, I reserve the right to supplement or amend my report as appropriate.

[1] Throughout this report, I use the term PFAS to refer to the group of thousands of different chemicals that includes PFOA, PFOS, and other per- and polyfluoroalkyl substances, which is consistent with the terminology used by US EPA (2020a) and ITRC (2020). Some scientists use the term "PFCs" or "perfluorinated chemicals" to refer to this group of chemicals, but "PFCs" is also used as an abbreviation for perfluorocarbons, which are a subset of perfluorinated chemicals that are closely related to PFAS but differ from PFAS in their structure, use, and toxicological and environmental properties (US EPA, 2020a). While both perfluorocarbons and PFAS molecules contain fluorine and carbon atoms, PFAS can also include oxygen, hydrogen, sulfur, and/or nitrogen atoms, whereas perfluorocarbons only contain carbon and fluorine atoms (US EPA, 2020a). Thus, according to US EPA (2020a), perfluorocarbons are distinct from PFAS.

[2] It is my opinion that the epidemiological, toxicological, and mode-of-action evidence do not demonstrate that humans have been harmed from PFAS exposure either in occupational or community studies (see, for example, ATSDR, 2018a; Australia, Expert Health Panel for PFAS, 2018; Michigan PFAS Science Advisory Panel, 2018), which is also consistent with the recent conclusions of investigators who evaluated the potential human health effects of PFOA as part of the C8 Science Panel (Steenland *et al*., 2020). Nonetheless, in this report, I discuss how risk-related parameters, such as exposure and dose, may vary among proposed class members for consideration in connection with the class certification process.

1.2 Qualifications of Dr. Barbara D. Beck

I received an A.B. degree (*cum laude*) in biology from Bryn Mawr College and a Ph.D. in molecular biology and microbiology from Tufts University. I am an expert in toxicology, with a specialty in human health risk assessment. I hold several certifications in toxicology, listed as follows. I am a diplomate of the American Board of Toxicology and a fellow and past president of the Academy of Toxicological Sciences. Both of these certifications are internationally recognized. The membership of each organization includes individuals from many countries, including countries in Europe, Asia, and Africa, as well as North America. I am also certified as a European Registered Toxicologist through the United Kingdom Registry of Toxicologists. All of these certifications are current. I was recently appointed as a fellow in the American Association for Advancement of Science.

I am a principal at Gradient, an environmental science consulting company that specializes in the fate and transport of chemicals in the environment and human health risk assessment.

From 1985 through 2018, I was a visiting scientist in the Department of Environmental Health at the Harvard T.H. Chan School of Public Health.

In addition, I was a research associate and a fellow in the Interdisciplinary Program in Health at the Harvard T.H. Chan School of Public Health, where I developed a short-term bioassay to predict the toxicity of particulate matter and gases for the lungs. I was also an editor and author of a monograph on variations in susceptibility to inhaled pollutants.

I was regional expert in toxicology and chief of the Air Toxics staff for Region I of the United States Environmental Protection Agency (US EPA), which includes the New England states. In this capacity, I provided advice on matters of toxicology, particularly as related to air toxics.

I joined Gradient in 1987. My consulting practice consists of health risk assessments for cancer and non-cancer endpoints, review of animal toxicology and human epidemiology studies, multi-media assessment of exposure to environmental chemicals, and evaluation of the historical development of toxicology.

I have served in an advisory capacity to a wide range of governmental and non-profit institutions on issues relating to toxicology, risk assessment, and public health. I have been active in the Society of Toxicology for many years, in both elected and appointed roles. In 2019, I was appointed to the US EPA Science Advisory Board (SAB). In 2020, I was appointed as vice chair of the US EPA SAB.

I have published articles on toxicology and risk assessment in peer-reviewed journals, books, and meeting proceedings. These publications have addressed a range of topics, such as the use of toxicology in the regulatory process and the toxicology of specific chemicals, such as metals and complex organic compounds. I have been a peer reviewer, which includes editorial positions, for multiple journals.

I have evaluated epidemiological, animal toxicological, and mechanistic studies for purposes of drawing conclusions regarding the risk to humans from exposure to specific chemicals. These analyses, which have been conducted using appropriate scientific principles and various analytical frameworks, have addressed a wide range of chemicals, including metals, chlorinated solvents, PFAS, and aromatic compounds.

I co-authored a manuscript on PFAS (Pizzurro *et al.*, 2019) and six studies of PFAS that were presented at the 47th, 55th, 57th, 58th, and 59th Annual Society of Toxicology meetings (Lewis *et al.*, 2008; Lynch *et al.*,

2016; Pizzurro *et al*., 2018; Seeley *et al*., 2018; Kerper *et al*., 2019; Boomhower *et al*., 2020) and have been designated as an expert on the toxicology of PFAS in several litigation cases in the US.

I have been a designated expert and given deposition testimony in cases involving 3M on five occasions: once in January 2007 (Felicia Palmer *et al*. *vs*. 3M Company; State of Minnesota, Tenth Judicial District, County of Washington, Case C2-04-6309), once in August 2008 (Gary A. Paulson, Karen Paulson, William Henry, and Bradley Krank *vs*. 3M Company; State of Minnesota, County of Washington, Case C2-04-6309), once in November 2017 (State of Minnesota *vs*. 3M Company; State of Minnesota, Fourth Judicial District, County of Hennepin, Civil File No. 27-CV-10-28862), once in January 2020 (Tommy Lindsey *et al*. *vs*. 3M Company *et al*.; US District Court, Northern District of Alabama, Case 5:15-cv-01750-AKK), and once in December 2020 (Michele Baker *et al*. *vs*. Saint-Gobain Performance Plastics Corp. *et al*.; US District Court, Northern District of New York, Case 1:16-CV-917). I was also a designated expert for 3M, but never testified, in the Matter of 3M United Kingdom PLC *vs.* The States of Guernsey *vs.* 3M Company, High Court of Justice, Queen's Bench Division, Commercial Court, Claim No. 2013 Folio 1044. In addition, at the request of 3M, I submitted comments on the US EPA draft Health Effect Documents for PFOA and perfluorooctane sulfonate (PFOS) in 2014, and on the National Toxicology Program draft systematic review of immunotoxicity associated with PFOA and PFOS in 2016. See Appendix B for a complete record of my testimony for the past four years.

2 Summary of Opinions

As a toxicologist and human health risk assessor, I have been asked to evaluate the extent to which the characteristics of the proposed class in the matter of Hardwick *vs*. 3M Company *et al*. may vary from an exposure, dose, and risk perspective with respect to the thousands of different types of PFAS that are the subject of the Plaintiff's claims. The Plaintiff alleges that exposures to various types of PFAS from various Defendants have resulted in contamination of his blood with PFAS and biopersistence and bioaccumulation of PFAS within his body, and has requested certification of a nationwide class. I considered the nature and extent of variability in parameters relating to potential risk,[3] such as exposure and dose, among proposed class members. My specific opinions are summarized below. To the extent that I receive additional materials, I reserve the right to update my conclusions as appropriate.

- The development of reliable conclusions regarding risks from chemical exposures entails considering the principles of toxicology, epidemiology, and risk assessment, including an evaluation of exposure, dose, and risk. For my analysis in this case, I evaluated variability in the factors that influence exposure to the many different types of PFAS by developing a conceptual model of the sources, transport, exposure media, and receptors (*i.e.*, humans who may be exposed to PFAS) of the various PFAS in the environment, and identifying the extent of variability in the underlying parameters in the model. I also evaluated variability in serum concentrations of PFAS across different populations, as well as variability in factors that potentially influence the toxicity of different types of PFAS. My analysis demonstrates that the magnitude of variability is such that individualized assessments would be required to perform a scientifically reliable assessment of exposure, dose, and risk of PFAS across proposed class members.
- PFAS are a broad category of organic molecules containing fluorine and carbon that consists of thousands of distinct chemicals. A subset of these compounds contains a chain of between 4 and 16 carbons ("C4" to "C16") that are fully bonded to fluorine atoms and another chemical group, such as a carboxylate or sulfonate group, that varies with different PFAS. The biological properties of individual PFAS depend on the particular chemical configuration of molecules and can vary by both carbon chain length and the particular functional groups that are present. Certain PFAS have been found to resist degradation in the environment and have the potential to accumulate in certain organisms. Nonetheless, such findings do not indicate that the presence of PFAS in the environment or in living organisms means that adverse effects are occurring or are likely to occur. Such a determination must be made in the context of other relevant information.
- My conceptual model of potential PFAS exposure pathways describes the variability in potential and suspected sources of PFAS that individuals in the US may be exposed to, as well as the potentially impacted exposure media in the environment, the exposure routes, and the exposed individuals or populations (*i.e.*, receptors). Given the many variables that affect the sources, amounts, and types of PFAS exposures, doses, and risks that any particular individual may have, it is not possible to reliably characterize an individual's potential risk (if any) from any particular PFAS (much less collectively) without examining those individual parameters. Details on the variability in the underlying parameters of the model are described below.
- Individual variability factors such as intake, body mass, age, gender, pregnancy status, and (in the case of certain PFAS) menstruation are some of the critical determinants of chemical dose and risk.

[3] Risk is the likelihood of an effect occurring, such as an adverse health effect from exposure to a chemical.

Intra-individual variability occurs from changes in an individual over time, such as physical (*e.g.*, body weight and age) and behavioral changes (*e.g.*, ingestion rates) (US EPA, 2019a). For example, lactating women are likely to ingest higher concentrations of PFAS from drinking water than non-lactating women due to their increased water intake rate needed to aid milk production (US EPA, 2016a,b). Interindividual variability, or differences among individuals within a population (*e.g.*, in factors such as age, gender, occupation, diet, race, behavior), may also impact PFAS intakes. Variability in these factors will result in variability in dose, indicating that the evaluation of potential health risks (if any) from exposure to PFAS requires assessment of intakes on an individualized basis.

- There is significant variability in serum concentrations of four example PFAS (PFOA, PFOS, perfluorohexane sulfonate [PFHxS], and perfluorononanoic acid [PFNA]) across and within the following populations: the general US population, worker populations exposed to PFAS during its manufacture or use, and populations of firefighters. Serum concentrations of these PFAS also vary by gender, pregnancy status, age, and race. This variability provides empirical support for the proposition that PFAS exposures are expected to vary among proposed class members, such that the evaluation of the risk of potential health effects (if any) from those exposures requires individualized assessment.
- The toxicokinetic processes of absorption, distribution, metabolism, and excretion (ADME) influence PFAS doses and responses in the body. These processes are variable across different PFAS, resulting in different elimination half-lives for different PFAS. These processes are also variable among individuals depending on pregnancy and breastfeeding status, menstruation, or underlying health conditions (*e.g.*, kidney disease), resulting in variability in serum concentrations for different PFAS and across sexes, doses, and life stages. This variability indicates that the evaluation of the potential health effects of exposure to PFAS (if any) would require assessment of the specific PFAS at issue, the exposure and dose for each PFAS, and, hence, the PFAS-specific risk, on an individualized basis.
- Different PFAS do not all share the same toxicological properties or target organs where effects are seen in experimental animal models. Accordingly, health-based criteria for particular PFAS from national and state agencies are based on different target organs and endpoints and have different values, both across agencies and across PFAS. The variability in target organs, endpoints, and toxicokinetic properties across different PFAS, as demonstrated in experimental animal models, indicates that all PFAS cannot be grouped together as a single class of chemical for the purpose of evaluating risk to individuals. In addition, there is no confirmed evidence in the literature to indicate that a combination of multiple different PFAS operates synergistically to cause adverse health effects in humans. Thus, a reliable characterization of risk (if any) for any particular individual from exposure to PFAS requires an assessment of the particular PFAS to which the individual was exposed, along with the exposure, dose, and risk parameters specific to that individual. These parameters will vary significantly across members of the Plaintiff's proposed class.

3 Methodology

3.1 Information Used and Analyses Performed

My opinions are based on my training and experience in toxicology and risk assessment and a review of the documents available as of the date of this report. Specific documents I have cited are listed in the References section. In addition, my opinions are informed by my extensive professional history and experience in toxicology and risk assessment. The types of information I relied upon for my analyses in this matter include the following:

- The complaint and motion for class certification specific to this case.
- General guidance documents in the fields of toxicology and risk assessment by agencies such as US EPA and the Agency for Toxic Substances and Disease Registry (ATSDR).
- Publicly available environmental and regulatory documents that are not case-specific but provide data and information that are relevant to my analyses. Such documents include toxicity criteria or guidelines and secondary toxicological or environmental reviews.
- Scientific literature, such as peer-reviewed journal articles and scientific textbooks that review toxicology and epidemiology principles, and toxicological and epidemiological studies relating to PFAS.

The specific analyses I performed include the following:

- I identified the key scientific principles relevant to evaluating the potential health risks of chemicals in the environment.
- I evaluated variability in the factors that influence exposure to the thousands of different PFAS by developing a conceptual model of the sources, transport, exposure media, and receptors of the various specific PFAS in the environment.
- I evaluated variability in serum concentrations of various PFAS in the general population, workers with known PFAS exposure, and firefighters.
- I evaluated variability in factors that influence the potential toxicity of different PFAS.

3.2 Evaluating Risks from Chemical Exposures

3.2.1 Introduction to Toxicology

An understanding of the scientific principles in the fields of toxicology and risk assessment is necessary for evaluating the potential for a relationship between exposure to chemicals and health effects. One of the most fundamental concepts in the field of toxicology is the dose-response relationship. Dose refers to the amount of a chemical taken into the body from different exposure pathways (*e.g.*, ingestion or inhalation). The dose-response concept is commonly summarized as "the dose makes the poison," which was first attributed to the famous Swiss-German physician-physicist Paracelsus in the 16th century (Aleksunes and

Eaton, 2019). Virtually all substances exhibit a dose-response relationship, which is characterized by response levels that increase as dose increases. However, for most chemicals, biological effects occur only when the dose exceeds a threshold level for a certain period of time. At doses between zero and the threshold, biochemical or physiological mechanisms can negate a chemical's effects, thereby preventing any adverse effects. As the magnitude and duration of exposure begin to exceed the threshold, these protective mechanisms can become less effective. Consequently, the effect begins to appear in a manner that corresponds to the increase in dose.

The specific effect will vary from chemical to chemical. For example, small amounts of salt may be consumed without adverse effects, because the body is able to adequately maintain proper salinity levels in its fluids and tissues. The adequate intake of salt needed to sustain health ranges from 3 to 3.8 g/day, depending on age, sex, and lifestyle (active or sedentary) (IOM, 2005). However, ingestion of much larger quantities of salt can override these homeostatic mechanisms and lead to adverse effects, such as hypertension (abnormally high blood pressure), in some individuals (IOM, 2005). Similarly, aspirin also shows a dose-response relationship. At the recommended dose of two tablets, aspirin provides pain relief from headaches or other minor aches. Even lower doses over extended durations can be beneficial in people with a history of cardiovascular disease (Burke *et al*., 2006; Ittaman *et al*., 2014). Taking more than the recommended dose, however, may eventually lead to toxicity (Roberts and Morrow, 2001; Burke *et al*., 2006). Ingestion of 10 aspirin tablets is associated with nausea, 30 tablets with acidosis (excess acidity in the blood) and hyperventilation, and 65 tablets with brain damage. Hemorrhage and death occur with ingestion of 100 tablets (Roberts and Morrow, 2001; Burke *et al*., 2006). Thus, because the appearance of biological effects is directly related to the magnitude and duration of an individual's exposure, estimation of dose is a critical element in determining potential human health risks from chemical exposures.

In some cases, an increased theoretical risk can be calculated based on a certain chemical dose even though the actual risk associated with that level of chemical exposure could be nonexistent or very small, as is the case with certain calculated risk levels compared to the risk of cancer from background environmental levels of a substance. For example, in studies investigating chronic (long-term) exposure to arsenic in soil, it is not until concentrations of arsenic in soil are approximately 100 mg/kg or higher that a consistent impact on body burden can be detected (Wong *et al*., 1992; Hewitt *et al*., 1995; Valberg *et al*., 1997; Gebel *et al*., 1998; Tollestrup *et al*., 2003; Tsuji *et al*., 2005), even though a hypothetical excess risk can be calculated at low concentrations of arsenic in soil.

Another important factor considered in toxicology is the frequency and duration of a chemical exposure. The period over which the dose of a chemical is received may be critically important in determining the resulting health effects (see, for example, US EPA, 1989; Paustenbach and Madl, 2014). For example, ingestion of sufficient quantities of ethanol (alcohol) in a single event may lead to central nervous system depression, coma, and death. However, lower doses, if repeated over years, may lead to liver and cardiovascular damage, effects not observed in acute alcohol intoxication (Fleming *et al*., 2006). At even lower doses, adverse effects will not occur. In fact, smaller doses (for example, a maximum of one to two glasses of red wine per day) may be beneficial, decreasing the risk of cardiovascular mortality (Goldberg *et al*., 2001; Harvard T.H. Chan School of Public Health, 2019). Thus, the severity of health effects can be different if the chemical exposure is experienced acutely in a single dose than if the chemical exposure is experienced chronically, with the same cumulative dose spread out over time. This is because during chronic exposure to the same cumulative dose, the body has time to eliminate the dose *via* excretion, to repair any damage that may have occurred, or to adapt and find other means of accommodating the chemical dose (Aleksunes and Eaton, 2019).

It should also be appreciated that not all of the changes observed in studies (in humans or animals) are adverse. Rather, the body is able to adapt to various chemical and physical stresses to maintain homeostasis. Only when the body's natural homeostatic and defense mechanisms become overwhelmed will the stress or

toxic insult lead to an adverse toxicological outcome. Reversibility is another concept that is important to consider when evaluating potential health effects (in particular for short-term exposures). For many exposures, when exposure to a chemical ceases, the health effect or symptom also ceases, with no long-term adverse consequences. For example, in animal experiments with PFAS, reversibility has been demonstrated for a variety of effects, including changes in liver effects, serum cholesterol, and thyroid hormone levels (see, for example, Perkins *et al*., 2004; Butenhoff *et al*., 2012).

3.2.2 Introduction to Epidemiology

Epidemiology has been defined as the study of the occurrence of disease or other health-related characteristics in human populations (Driscoll and Winder, 2004). Epidemiology studies measure actual disease outcomes in a population and examine associations that may exist between chemicals and adverse health effects. It is common for data from epidemiological investigations to be used in risk assessment, a tool to predict adverse health effects based on knowledge of the effects of chemicals and exposures (US EPA, 1989; Faustman, 2019). A major strength of epidemiology studies is that they deal with humans and real exposures (Olsen *et al*., 2014). Unlike scientists involved in laboratory investigations, however, epidemiologists are rarely able to exert control over the parameters of their studies and must grapple with a variety of technical biases and confounding variables (Driscoll and Winder, 2004; Olsen *et al*., 2014). As a result, robust exposure estimates are often difficult to obtain from epidemiology studies, because they are frequently done retrospectively (*e.g.*, through employment records). Another challenge of interpreting epidemiology studies is that subjects are often exposed to multiple chemicals, especially when a lifetime exposure period is considered (Faustman, 2019).

There are different types of epidemiology studies, each with different strengths and weaknesses. Two major design types are cohort and case-control studies (Faustman, 2019). In a cohort study, subjects are selected based on their exposure status (exposed *versus* non-exposed), then the proportion of each group that gets the disease or condition of interest is evaluated. Cohort studies are useful when the exposure of interest is rare (Hennekens *et al*., 1987). In case-control studies, the case group consists of subjects who have the disease of interest, while the control group consists of those who do not. The groups are then compared with respect to the proportion of each that has a history of the exposure of interest. Case-control studies are commonly used for studying rare diseases or diseases with a long latency (Hennekens *et al*., 1987). Longitudinal studies, such as prospective cohorts, that follow the exposure of a population over time are more useful for assessment of causal associations than cross-sectional studies that measure disease and concurrent exposure at only one point in time (Faustman, 2019).

3.2.3 Toxicity Guidelines

For chronic (*i.e.*, long-term exposure) non-cancer effects, toxicity is typically characterized using a chemical-specific reference dose (RfD) for ingested chemicals or reference concentration (RfC) for inhaled chemicals. For cancer, toxicity is typically characterized using a cancer slope factor (CSF) or inhalation unit risk (IUR).

The RfD or RfC is an estimate (with uncertainty spanning perhaps an order of magnitude or more) of a daily oral exposure or continuous inhalation exposure, respectively, of a human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (Barnes and Dourson, 1988; US EPA, 2011a). RfDs and RfCs are derived from a no observed adverse effect level (NOAEL) or a lowest observed adverse effect level (LOAEL). NOAELs and/or LOAELs are typically identified from studies in laboratory animals, most often rats or mice, in which animals are exposed *via* ingestion or gavage (*i.e.*, oral exposure). To account for potential uncertainties and variability (differences between laboratory animals and humans, variation in sensitivity among individuals, use of a

LOAEL instead of a NOAEL, insufficient study duration, missing key toxicological studies, *etc.*), the NOAEL is divided by uncertainty factors (UFs), typically factors of 10 each, to account for inter- and intraspecies variability and, if necessary, other sources of variability (US EPA, 2002).[4] The application of UFs helps ensure that exposures at or below the RfD or RfC are associated with negligible, if any, risk, even in sensitive populations. However, adverse health effects will not necessarily occur even at exposures greater than the RfD or RfC.

As described by US EPA (2000):

> It should be noted that exposures above an RfD or RfC do not necessarily imply unacceptable risk or that adverse health effects are expected. Because of the inherent conservatism of the RfC/RfD methodology, the significance of exceedances must be evaluated on a case by case basis, considering such factors as the confidence level of the assessment, the size of UFs used, the slope of the dose-response curve, the magnitude of the exceedance, and the number or types of people exposed at various levels above the RfD or RfC.

In 2014, US EPA reiterated how values in its Integrated Risk Information System, including RfDs and RfCs, "cannot be validly used to accurately predict the incidence of human disease or the type of effects that chemical exposures have on humans. This is due to the numerous uncertainties involved in risk assessment, including those associated with extrapolations from animal data to humans and from high experimental doses to lower environmental exposures" (US EPA, 2019b).

For cancer, as part of a regulatory risk assessment, US EPA frequently uses mathematical models to extrapolate risks observed at high doses or concentrations (either in humans or animals) down to low doses/concentrations typical of actual exposure levels, usually based on the conservative assumption that there is no threshold for cancer.[5] Using these models, either a CSF or IUR is identified, which represents the incremental (*i.e.*, above background) upper-bound risk of an additional cancer per dose (in mg/kg-day) or concentration (in $\mu g/m^3$) of the chemical, assuming low-dose linearity (US EPA, 2005). US EPA defines the IUR as the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 $\mu g/m^3$ in air (US EPA, 2005).

Because the CSF or IUR is an upper-bound value, US EPA recognizes that actual (or "true") cancer risks would likely be lower than those calculated using the CSF/IUR (US EPA, 2005). US EPA indicates that the "use of upper bounds generally is considered to be a health-protective approach for covering the risk to susceptible individuals" (US EPA, 2005).

Assuming linearity at low doses is a conservative assumption that is used as a default when chemical-specific information regarding the shape of the dose-response curve at low doses is uncertain. Based on the current understanding of the carcinogenic process, many scientists have concluded that this model is likely to overestimate risks for many chemicals (see, for example, US EPA, 2005; Cohen and Arnold, 2011; Aschner *et al*., 2016; Boobis *et al*., 2016; Cohen, 2017).

[4] There is a role for scientific judgment in the selection of UFs and this can lead to differences across agencies in the resulting guidelines for the same chemical.

[5] It should be noted that this is a regulatory construct and not a reflection of "true" risks. That is, regulatory risk estimates are protective, but not predictive, and the true risk is likely to be well below the hypothetical regulatory risk estimate. See, for example, James *et al*. (2015).

3.2.4 Exposure Assessment

The primary objectives of exposure assessment are to determine the source, type, magnitude, and duration of an individual's or group's contact with a specific chemical or chemicals of interest (Federal Judicial Center and NRC, 2011; Faustman, 2019). Exposure assessments can address past, current, or future exposures (Federal Judicial Center and NRC, 2011; Paustenbach and Madl, 2014) and involve identifying and evaluating various exposure pathways relevant to the site in question.

An exposure pathway is the link between environmental releases and populations that might come into contact with or be exposed to environmental contaminants. A complete exposure pathway involves five elements (ATSDR, 2005). First, there has to be a contaminant source or release; this is the origin of the environmental contamination. The second element is environmental fate and transport. Once released to the environment, contaminants may move through and across different media (*e.g.*, air, groundwater) and some may degrade as they do so. Evaluating environmental fate and transport involves identifying all environmental media that may serve to transport contaminants from their source(s) to possible points of human exposure. Third, there needs to be an exposure point or area where people might come into contact with chemicals in a given medium. For example, an exposure area could be a surface water body used for recreational purposes. It should be noted that exposure points or areas may change over time with changes in land use. Fourth, there needs to be an exposure route; that is, a means by which people physically come into contact with environmental contamination at the exposure point or area. Exposure routes are typically inhalation (*i.e.*, of contaminants in air), ingestion (*e.g.*, of contaminants in food), and dermal contact (*e.g.*, with contaminants in sediment). Fifth, there needs to be a potentially exposed population; that is, a population (*e.g.*, resident) who comes into contact with contaminants.

Overall, the presence of a complete exposure pathway will require a source and mechanism of contaminant release to the environment, an environmental transport medium, a point or area of potential contact between a receptor and the environmental medium, a feasible exposure route at the contact point or area, and a potentially exposed population (the receptor or receptors). If any one of these elements is missing, the exposure pathway is not considered complete. For example, if human activity patterns and/or the location of potentially exposed individuals relative to the location of the affected media prevent individuals from coming into contact with that media, then that exposure pathway is not complete.

3.2.5 Risk Assessment

Risk assessment is defined as "the characterization of the potential adverse health effects of human exposures to environmental hazards" (NRC, 1983). Risk assessments form the scientific basis for many regulations or guidelines pertaining to environmental contaminants. These include maximum contaminant levels for contaminants in drinking water, National Ambient Air Quality Standards for criteria air pollutants, Maximum Achievable Control Technology standards for hazardous pollutants, emissions standards for hazardous mobile source pollutants, and limits on the use of pesticides (NRC, 2009).

Risk assessment involves the following four steps, which were first presented as a framework by the National Academy of Sciences in 1983 (NRC, 1983):

1. **Hazard Identification:** The potential hazard is identified; this involves determining whether a particular chemical is causally linked to health effects.
2. **Dose-Response Assessment:** A dose-response assessment is performed to determine the relationship between the magnitude of exposure to the hazard and the probability of the occurrence of a health effect.

3. **Exposure Assessment:** The level of human exposure to the hazard is estimated.
4. **Risk Characterization:** The estimated exposure level is compared with the value obtained from the dose-response assessment and characterized in a risk estimate, with an assessment of the magnitude of uncertainty.

Toxicologists like myself frequently rely on this four-step methodology to guide our analyses of potential human health risks in a reliable manner. In some situations, a screening risk assessment is employed before the decision to conduct a complete risk assessment is made. US EPA has published a set of risk-based screening values for various media, including residential and industrial soil, air, and drinking water, known as Regional Screening Levels (RSLs) (US EPA, 2019c) for use in these types of evaluations. US EPA derives RSLs by combining generic conservative exposure assumptions with US EPA toxicity criteria. These levels are considered to be protective for humans (including sensitive subgroups) over a lifetime (US EPA, 2020b). They are not intended to (and do not) indicate levels at which human health effects can or will occur.

RSLs are protective of human health in hypothetical high-end (*e.g.*, intentionally using assumptions that overestimate chemical intake), chronic exposure scenarios. RSLs are intentionally conservative to help identify sites that do not warrant further investigation (*i.e.*, when all maximum detected constituent concentrations are less than corresponding RSLs). As described under US EPA guidelines, screening values are used to identify chemicals of interest at a site for the purposes of further investigation and decision making (US EPA, 2020b). RSLs are meant to be used as conservative comparison values for screening site concentrations, not as final cleanup standards; the goal of screening is to determine areas and contaminants that require further evaluation. Exceedance of a screening level does not mean that an exposure presents an unacceptable health risk, only that further evaluation of potential risks is warranted (US EPA, 2020b).

3.2.6 Regulatory Toxicology *vs*. Causation Analysis

There are substantial differences in how toxicological data are used in a regulatory framework to protect public health *versus* how those data are used to make determinations regarding general causation, *i.e.*, whether a chemical is established as a cause of disease in humans, in a situation in which individuals are claiming various health symptoms from chemical exposure (Marchant, 2019) and assessment of individual health risk is needed. The approach to regulatory decision making is, in part, directed by policy. As practitioners of public health, regulatory toxicologists are concerned more with avoiding potential adverse health effects, even in the face of uncertainty, than with estimating the likelihood of health effects actually occurring in a population or an individual (US EPA, 2004; ATSDR, 2018b). This difference in perspective is important, because regulators often use high-end estimates of exposure and toxicity (which can result in overprediction of potential health risks) to be protective of human health. The aim of US EPA and other public health agencies is not to precisely define which effects are possible or expected to occur, but to define a level at which health effects are *unlikely* to occur (US EPA, 1993; ATSDR, 2018b). Thus, regulatory criteria are designed to "protect the health of everyone in general and no one in particular" (Rodricks and Rieth, 1998). Indeed, US EPA and other agency guidelines for developing regulatory criteria state that such criteria are applicable to "susceptible groups" or sensitive subpopulations, which include life stages (such as developing individuals [embryo, fetus]) and other factors that may predispose individuals to greater response to an exposure (US EPA, 2002; CalOEHHA, 2008). In some cases, regulatory agencies are required by statute to apply these highly conservative safety factors; for instance, the Food Quality Protection Act requires that an additional 10-fold safety factor be accounted for when evaluating pre- and postnatal pesticide exposures (US EPA, 2016c).

In contrast to evaluations performed for regulatory or guidance purposes, assessing the risk of disease in an individual from a specific chemical exposure (*i.e.*, a toxicological causation analysis) requires an estimate of *actual* risk, based on an individual exposure assessment, dose characterization, and an understanding of what health effects, if any, have been demonstrated in humans from the particular chemical at issue (Olsen *et al*., 2014).

In this type of analysis, a lack of exceedance of a risk-based screening level can be used as an exclusionary tool (*i.e.*, to remove a chemical or pathway from further consideration). And, for the reasons stated above, it is scientifically inappropriate to interpret an exceedance as indicative of a causal link between the chemical and the observed health effect. In sum, regulatory levels do not set a scientific dividing line between "safe" and "unsafe" exposures, and one cannot conclude that any exposure to levels above regulatory guidance or limits establishes that any health effect was caused or may be caused in the future by that exposure.

4 Overview of PFAS Chemistry and Properties

4.1 The Chemistry of PFAS

PFAS are a broad category of organic molecules containing fluorine and carbon that consists of thousands of distinct chemicals. A subset of these chemicals contains a chain of between 4 and 16 carbons that are fully bonded to fluorine atoms and another chemical group, such as a carboxylate or sulfonate group, that varies with different PFAS (ATSDR, 2018a; US EPA, 2020a). Several PFAS have either a sulfonate group or a carboxylate group attached to a terminal carbon, and the structures of some examples of these PFAS are depicted in Figure 4.1.

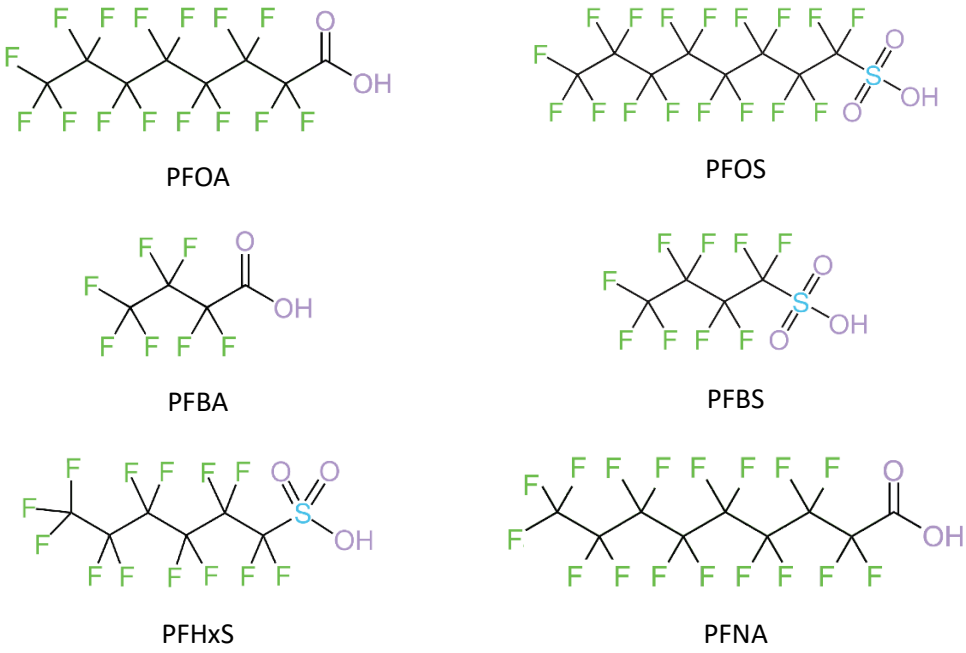


Figure 4.1 Chemical Structures of PFOA, PFOS, PFBA, PFBS, PFHxS, and PFNA. PFBA = Perfluorobutanoic Acid; PFBS = Perfluorobutane Sulfonate; PFHxS = Perfluorohexane Sulfonate; PFNA = Perfluorononanoic Acid; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctane Sulfonate.

PFAS are produced by two major methods, telomerization and electrochemical fluorination (ATSDR, 2018a; ITRC, 2020). Depending on the manufacturing process, many PFAS may occur as mixtures of linear and branched isomers in different proportions. The electrochemical fluorination process produces mixtures of linear and branched isomers, whereas the telomerization method produces mainly linear isomers. These structural differences may affect the biological properties of a particular PFAS (ATSDR, 2018a).

PFAS do not occur naturally and are the result of anthropogenic activities (ATSDR, 2018a).[6] Historically, some manufacturers used PFOA as a processing aid in the creation of fluoropolymers that have the ability to repel oil, stains, grease, and water under high temperatures, making these chemicals ideal for use in making protective coatings for non-stick cookware and clothing (Lau, 2015). PFOS possesses surfactant properties (the ability to reduce the surface tension between liquids or between a liquid and solid). Because of these properties, PFOS or related chemistries have been used in numerous chemical applications, such as firefighting foams, hydraulic fluids, carpet cleaners, and oil well surfactants. Tables 4.1 and 4.2 present a small subset of sulfonated and carboxylated PFAS, respectively, described in ATSDR (2018a) and/or Dagnino (2015).

Table 4.1 PFAS with a Sulfonate Functional Group

Compound	Abbreviation	Carbon Length
Perfluorobutane Sulfonate	PFBS	4
Perfluorohexane Sulfonate	PFHxS	6
Perfluorooctane Sulfonate	PFOS	8
Perfluorodecane Sulfonate	PFDS	10

Note:
PFAS = Perfluoroalkyl Substances.

Table 4.2 PFAS with a Carboxylate Functional Group

Compound	Abbreviation	Carbon Length
Perfluorobutanoic Acid	PFBA	4
Perfluoropentanoic Acid	PFPeA	5
Perfluorohexanoic Acid	PFHxA	6
Perfluoroheptanoic Acid	PFHpA	7
Perfluorooctanoic Acid	PFOA	8
Perfluorononanoic Acid	PFNA	9
Perfluorodecanoic Acid	PFDA	10
Perfluoroundecanoic Acid	PFUnA	11
Perfluorododecanoic Acid	PFDoA	12
Perfluorotetradecanoic Acid	PFTA	14

Note:
PFAS = Perfluoroalkyl Substances.

4.2 Environmental and Biological Properties of Different PFAS

Certain PFAS are resistant to degradation by water, sunlight, microbes, and animal metabolism. This resistance is attributable to the strong carbon-fluorine bond present in fully fluorinated PFAS, which makes these molecules very stable and non-reactive (ATSDR, 2018a). In addition, it has been shown that in certain circumstances, certain PFAS have the potential to enter the food chain, bioaccumulate in certain organisms, and undergo long-range transport away from the original source (ATSDR, 2018a). However, these properties vary significantly across different PFAS. One study detected PFOS in the tissues of fish, birds, and marine mammals in both urban and non-urban areas, with higher concentrations in predatory animals, suggesting that both transport of PFOS far from its anthropogenic sources and biomagnification of PFOS up the food chain are possible (Giesy and Kannan, 2001). In the same study, all concentrations of PFHxS were less than the limit of quantitation (LOQ), and only a few samples contained PFOA at levels greater

[6] Although it is commonly stated in the scientific literature that PFAS result only from anthropogenic activities, there is evidence of a PFAS occurring naturally in the environment (Von Sydow *et al*., 2000; Frank *et al*., 2002; Scott *et al*., 2005).

than the LOQ. In general, PFOS is the most commonly detected perfluorinated sulfonate in wildlife (Conder *et al*., 2008; ATSDR, 2018a).

The biological properties of a particular PFAS can depend on both carbon chain length and the particular functional groups that are present, as well as the isomer type (branched *vs.* linear). Certain PFAS with longer chain lengths, for example with eight carbons, have long biological half-lives in the body and can bioaccumulate under certain circumstances (Fujii *et al*., 2015; Lau, 2015; Buck, 2015; ATSDR, 2018a). Bioaccumulation potential decreases at chain lengths greater than eight (ATSDR, 2018a), likely due to a decreased capacity for absorption. In addition, certain sulfonate PFAS have longer half-lives than carboxylic acid PFAS of the same chain length (Lau, 2015; ATSDR, 2018a). Variation in chain length and functional groups also produces different health effects in experimental animals (ATSDR, 2018a).

5 Variability in Factors that Influence Exposure

In this section, I evaluate the variability in factors that influence exposure to PFAS, including the various sources of PFAS, their transport through different types of exposure media, and the exposed populations of individuals associated with different exposure pathways. Quantifying PFAS exposures is particularly challenging due to the multitude of PFAS compounds in various sources, which amplifies the variability in potential exposures. Understanding exposure variability is key to understanding the likelihood that an individual or group of individuals may be at elevated risk from a chemical exposure (Paustenbach and Madl, 2014; US EPA, 2011b, 2019a). Although it is my opinion that the overall epidemiological, toxicological, and mode-of-action evidence does not demonstrate that humans have been harmed from PFAS exposure, either in occupational or community studies (see, for example, ATSDR, 2018a; Michigan PFAS Science Advisory Panel, 2018; Australia, Expert Health Panel for PFAS, 2018; Steenland *et al.*, 2020), I understand that considering variability among individuals is part of the class certification process. It is clear that PFAS exposures are expected to be highly variable among the potential class members. Accordingly, even assuming that there is some dose at which a health effect might occur in humans, the evaluation of potential health risks (if any) from exposure to PFAS requires assessment of exposure on an individual level.

5.1 Conceptual Model

As noted in Section 3.2.4 and presented in Figure 5.1, a complete exposure pathway for a chemical involves a source or release of the chemical, environmental fate and transport, exposure point or area, exposure route, and exposed population. Multiple variables along each step of the exposure pathway can result in various exposure scenarios. However, exposure only occurs when the exposure pathway is complete and a chemical released from a source enters an individual receptor (US EPA, 2019a).

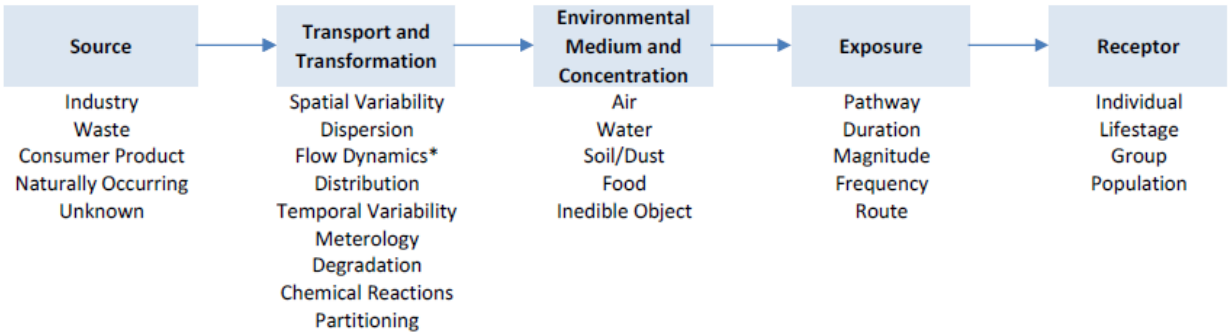


Figure 5.1 General Processes and Factors that Influence a Complete Exposure Pathway (adapted from US EPA's Guidelines for Human Exposure Assessment [US EPA, 2019a])

A conceptual model is a tool that maps out the theoretical links between all potential and suspected sources and anticipated receptors based on knowledge of the different steps that can result in exposure. US EPA (2016a,b) and the Interstate Technology and Regulatory Council (ITRC) (2020) have developed conceptual models outlining potential PFAS exposures based on various identified sources. I developed a conceptual model of potential PFAS exposure pathways for an individual (Figure 5.2). For this model, I defined

transport pathways from a PFAS source to an individual as either direct or indirect pathways. Direct pathways are those in which PFAS are transported directly from one step in the exposure process to the next (*e.g.*, a manufacturing plant releasing PFAS into the air *via* plant emissions, a resident drinking water containing PFAS). Conversely, indirect pathways involve additional fate and transport mechanisms that help transport PFAS from one step of the exposure process to the next (*e.g.,* some PFAS released into the soil may mobilize into the underlying aquifer and impact groundwater, and an individual is exposed to PFAS from consuming a homegrown produce watered with groundwater containing PFAS). The sources, pathways, media, and receptors in the conceptual model are described in more detail below. As illustrated in Figure 5.2, for each individual PFAS, there are many variables that will affect what any given individual's exposure to that PFAS will be over his or her lifetime. And, in light of those variables, each individual will have a unique mixture of PFAS exposures from a wide variety of different PFAS sources over his or her lifetime.

Source	Impacted Medium	Primary Exposure Medium	Secondary/Tertiary Exposure Medium	Receptor	Exposure Pathways: Ingestion	Dermal Contact	Inhalation
Waste material							
in Landfills	Neighboring groundwater	Drinking water	Produce	Residents	X	X	X
				Consumers	X	O	O
		Drinking water		Residents	X	X	X
		Irrigation water	Produce	Consumers	X	O	O
	Neighboring surface water bodies		Livestock/Game	Consumers	X	O	O
		Surface Water		Recreators	Incidental	X	X
			Fish/Shellfish	Consumers	X	O	O
		Sediment		Recreators	Incidental	O	O
	Air	Air		Residents	O	O	X
in Wastewater Treatment Plants (WWTP)	Receiving surface water bodies	Drinking water	Produce	Residents	X	X	X
				Consumers	X	O	O
				Recreators	Incidental	X	X
		Surface Water	Livestock/Game	Consumers	X	O	O
			Fish/Shellfish	Consumers	X	O	O
		Sediment		Recreators	Incidental	O	O
	Air	Air		Residents	O	O	X
in biosolids (used for agriculture fertilizer)	Soils where biosolids are applied	Soil		Workers	Incidental	X	X
				Consumers	X	O	O
		Produce	Livestock/Game	Consumers	X	O	O
Incineration of Waste	Air	Air		Gen. population	O	O	X
		Soil		Gen. population	Incidental	X	X
			Produce	Consumers	X	O	O
		Groundwater	Drinking Water	Gen. population	X	X	X
		Surface Water		Recreators	Incidental	X	X
			Fish/Shellfish	Consumers	X	O	O
Consumer Product							
in packaging (e.g. popcorn bags, fast food containers, pizza boxes)	Consumer Product	Packaging		Consumers	Incidental	X	X
		Food		Consumers	X	O	O
in building materials (e.g., composite wood, concrete, cable, wiring)	Consumer Product	Consumer Product		Consumers	Incidental	X	X
		House dust		Consumers	Incidental	O	X
in home use (e.g., non-stick cookware)	Consumer Product	Consumer Product		Consumers	Incidental	X	X
in home maintenance use (e.g., cleaners, paints, varnish, floor polish,	Consumer Product	Consumer Product		Consumers	Incidental	X	X
		House dust		Consumers	Incidental	O	X
in personal care product use (e.g., cosmetics, sunscreen, shampoo; dental floss/tape)	Consumer Product	Consumer Product		Consumers	Incidental	X	X
in apparel and textiles (e.g., clothing, furniture, carpet, shower curtains, footwear)	Consumer Product	Consumer Product		Consumers	Incidental	X	X
other (e.g., used in car materials, ski wax, paper)	Consumer Product	Consumer Product		Consumers	Incidental	X	X

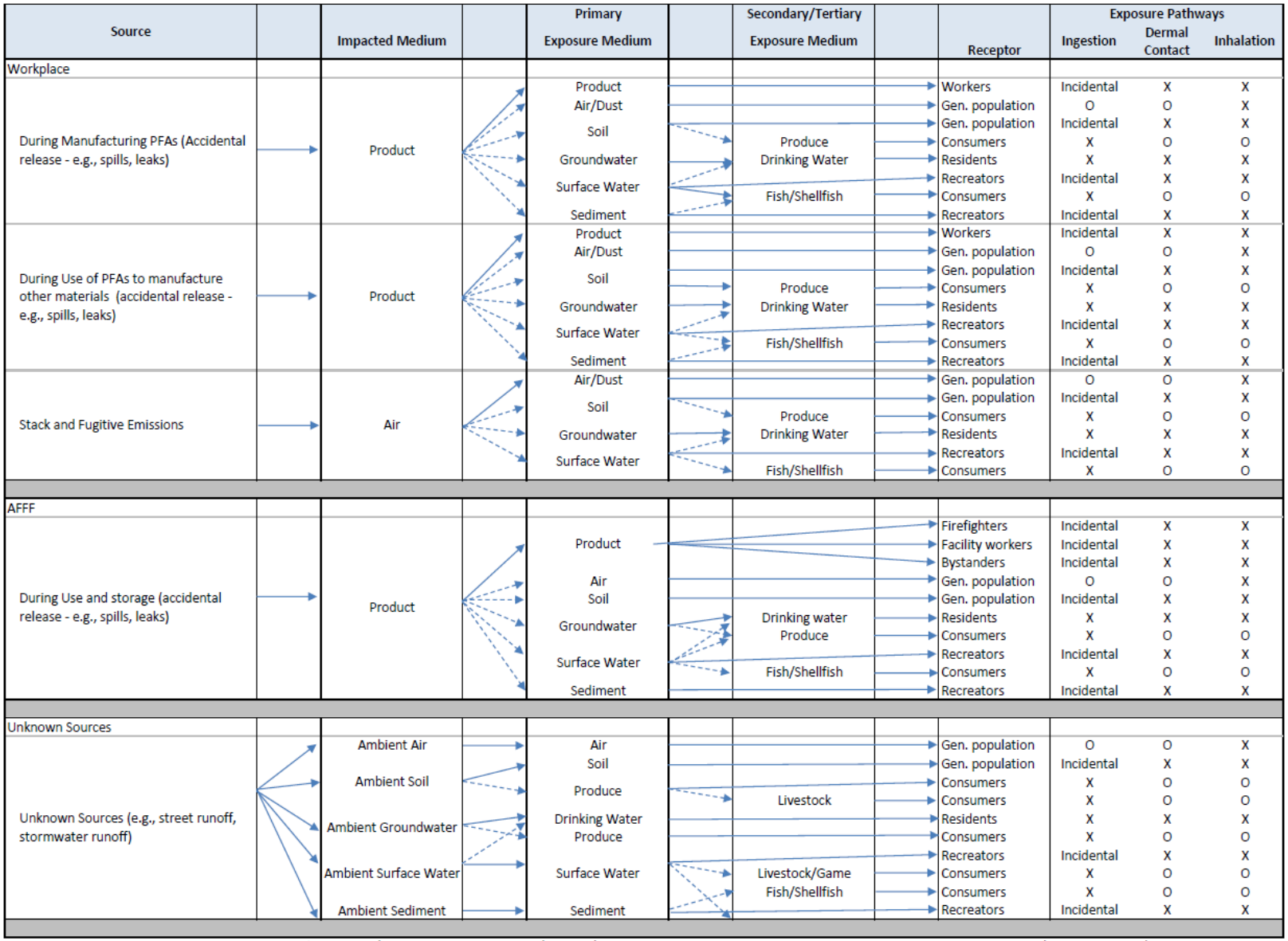


Figure 5.2 Potential PFAS Exposure Pathways

Once a particular PFAS is released into the environment, an individual may be exposed to that PFAS *via* an exposure pathway. I categorized exposure pathways as complete or incomplete exposure pathways for a given receptor based on three possible exposure routes:

- Ingestion – exposure occurs if an individual introduces PFAS into the gastrointestinal tract when consuming food, drinking, or inadvertently swallowing soil, dust, or foreign objects.
- Dermal contact – exposure occurs if PFAS are dermally absorbed through the skin upon contact.
- Inhalation – exposure occurs if an individual breathes PFAS as an aerosol or respirable particle.

A complete exposure pathway does not indicate risk, as other components such as concentration, contact rate, frequency, and duration can impact the intake (amount of chemical entering the body), dose (amount of chemical available for metabolic processes), and dose-response relationship (toxicity).

5.2 PFAS Sources

PFAS are man-made compounds that do not occur naturally in the environment (ATSDR, 2018a).[7] Certain of these PFAS have been produced since the mid-20th century and used extensively in a myriad of products and manufacturing processes due to their surfactant properties, stability, non-reactivity, and oil-, water-, stain-, and soil- repellent capabilities (ATSDR, 2018a; ITRC, 2020; Michigan PFAS Science Advisory Panel, 2018; Buck *et al*., 2011).

For the conceptual model (Figure 5.2), I grouped PFAS sources into the following five main categories: (1) waste materials; (2) consumer products; (3) workplace (4) aqueous film-forming foam (AFFF); and (5) unknown point sources. PFAS types, compositions, and concentrations will vary based on the source. Even within a given source, the formulation may vary widely based on a number of factors such as manufacturer, processes, use, or age of the waste or product, resulting in variability in exposures among individuals even to the same source. Thus, risks (if any) will vary based on the different sources of PFAS to which an individual may be exposed, requiring an individualized assessment.

5.2.1 Waste Materials

Pathways by which PFAS that have been used in industry and consumer products enter waste streams include landfills and wastewater treatment plants (WWTPs) (ATSDR, 2018a). Landfills are identified as a PFAS source (*via* landfill leachates) because they are an ultimate repository for industrial and municipal PFAS-contaminated wastes (ITRC, 2020). PFAS composition in leachate varies widely depending on the type of PFAS, the waste type, the age of the waste, and the climate of the area. For example, 5:3 fluorotelomer carboxylic acid (FTCA) is commonly found in municipal landfills where PFAS-containing carpets are disposed (ITRC, 2020). Leachate from municipal landfills within six cities across the Southern US detected variable concentrations of PFOS (329-53,100 ng/L), PFOA (939-48,100 ng/L), and perfluoroalkyl sulfonamide (FOSA) (249-258 ng/L) (3M, 2001). The highest concentrations were associated with a city where a fluorochemical manufacturing plant historically used the municipal landfill to dispose of industrial wastewater sludge (3M, 2001). Other PFAS such as n:2 fluorotelomer sulfonic acids, fluorotelomer unsaturated carboxylic acids (FTUCAs), and FOSA have also been detected in landfill leachate to varying degrees (Buck *et al*., 2011).

[7] As noted earlier, there is some evidence of a PFAS occurring naturally in the environment (Von Sydow *et al*., 2000; Frank *et al*., 2002; Scott *et al*., 2005).

WWTPs have been identified as point sources of PFAS from effluent releases into receiving water bodies, unintended releases from surface impoundments within the WWTP, and the application of WWTP biosolids (treated sewage sludge) (ITRC, 2020). WWTPs receive industrial wastewater, municipal wastewater, landfill leachate, and firefighting foam, all of which may contain various types of PFAS. Similar to landfill leachate, the PFAS composition of WWTP effluent varies depending on the specific PFAS, the waste type and transport, and the transformation processes (*e.g*., biological and chemical transformation and the physical or chemical partitioning) of the PFAS present in the wastewater (ITRC, 2020). Certain studies evaluating WWTP effluent samples found predominantly PFOS and PFOA, with lower concentrations of certain other PFAS (perfluorohexane sulfonate [PFHS], PFHxS, perfluorobutane sulfonate [PFBS], PFNA, perfluorodecanoic acid [PFDA], perfluoroundecanoic acid [PFUnDA], perfluorooctane sulfonamide [PFOSA], 8:2 FTCA, 8:2 FTUCA, FOSA, perfluoroalkyl sulfinic acid [PFSiA], perfluoroalkyl phosphinic acid [PFPiA], fluorotelomer phosphate monoester [monoPAP], and polyfluoroalkyl phosphoric acid diester [diPAPs]) (Sinclair and Kannan, 2006; Loganathan *et al*., 2007; 3M, 2001; Clarke and Smith, 2011; ATSDR, 2018a; ITRC, 2020; 3M, 2001; Buck *et al*., 2011). For example, Sinclair and Kannan (2006) analyzed effluent from six WWTPs and found elevated concentrations of PFOA and PFOS (8-1,050 ng/L and 3-68 ng/L, respectively) and lower concentrations of PFHS (<2.5-39 ng/L), PFNA (<10-376 ng/L), PFDA (<2.5-47 ng/L), PFUnDA (<2.5-10 ng/L), 8:2 FTCA (<2.5-7 ng/L), and 8:2 FTUCA (<2.5-29 ng/L). Biosolids from WWTPs are often used as fertilizer for agriculture and have a similar PFAS composition as the WWTP effluent, with higher concentrations of PFOS and PFOA and lower concentrations of certain other PFAS (Sinclair and Kannan, 2006; Loganathan *et al*., 2007; 3M, 2001; Clarke and Smith, 2011; ATSDR, 2018a; US EPA, 2016a). However, the individual PFAS composition can vary significantly across different WWTPs, given the variety of sources that may feed into the WWTP (Sinclair and Kannan, 2006; Loganathan *et al*., 2007; ATSDR, 2018a).

5.2.2 Consumer Products

PFAS have been widely used in the processes for making various consumer products. I divided PFAS detected in consumer products into seven broad categories: (1) food packaging materials; (2) building materials (3) home use products; (4) home maintenance products; (5) personal care products (6) apparel and textiles; and (7) other miscellaneous products, similar to the categories described in ITRC (2020), Trudel *et al*. (2008), and Washburn *et al*. (2005). With approximately 5,000-10,000 different PFAS manufactured (ITRC, 2020), each with specific properties and characteristics, the specific PFAS and the concentration used in each of these categories of consumer products vary. Furthermore, some of the PFAS formulas used in consumer products have been replaced with different formulas, including precursors that can be biotransformed to other PFAS in the body, increasing the variability in the types and concentrations of PFAS to which consumers may be exposed (Sunderland, *et al*., 2019). A description of each group of consumer products and examples of PFAS found in or related to these products are discussed below.

5.2.2.1 Food Packaging Materials

Over time, different PFAS have been used in the surface treatment of an array of food packaging materials such as popcorn bags, candy wrappers, fast food containers, paper cups, parchment paper, and pizza boxes, due to their oil- and water-resistant properties (Wang *et al*., 2013; Trudel *et al*., 2008; US EPA, 2016a,b; ITRC, 2020). The US Food and Drug Administration (US FDA) approved more than 90 unique monomer and polymer PFAS in food contact materials (ITRC, 2020). The most common PFAS detected in fast food wrappers include perfluoroalkyl carboxylic acids (PFCAs; *e.g*., PFOA and perfluorohexanoic acid [PFHxA]), perfluoroalkane sulfonic acids (PFSAs; *e.g*., PFBS), and fluorotelomer sulfonates (*e.g*., 6:2 FTS) (ITRC, 2020; Buck *et al*., 2011). Studies have also detected perfluoroalkyl carboxylates (*e.g*., PFOA and PFHxA), perfluoroalkyl sulfonates (*e.g*., PFBS), fluorotelomer sulfonates, and unknown polyfluorinated compounds in fast food packaging materials (ATSDR, 2018a).

5.2.2.2 Building Materials

Numerous different PFAS have been applied to building materials for their chemical properties designed to enhance the performance of these materials. Fluoropolymers such as fluorinated ethylene propylene (FEP), perfluoroalkoxy (PFA), and polytetrafluoroethylene (PTFE) have been applied to electrical wire casing and fire- and chemical-resistant tubing because of their heat-resistant properties (ATSDR, 2018a; ITRC, 2020). PFCA and PFOA have been applied to composite wood for additional product strength and durability, while perfluoroheptanoic acid (PFHpA) and other 5- to 8-chain PFCAs have been used in wood fiber insulation to enhance the performance at a lower cost (ITRC, 2020). PFOS has been used in phenolic foam building insulation and various PFAS (predominantly C8-C20 gamma-omega-perfluorotelomer thiols with acrylamide) have been used in concrete (ITRC, 2020).

5.2.2.3 Home Use Products

Various PFAS have been used in manufacturing processes to create a protective barrier in household items such as non-stick cookware, paper plates, and liners for ovens and trays (ATSDR, 2018a; ITRC, 2020).

5.2.2.4 Home Maintenance Products

Numerous different PFAS have been detected in home maintenance products such as cleaners, paints, varnish, floor polish, and insecticides (US EPA, 2016a). Short-chained sulfonamides are commonly used in herbicides, and PFAS such as N-ethyl perfluorooctane sulfonamide (N-EtFOSA), perfluorophosphonic acids (PFPAs), and PFPiAs are found in pesticides as active ingredients or anti-foaming agents (ITRC, 2020). PFOS-related chemicals are used in paints and varnishes (ITRC, 2020) and high levels of fluorotelomer alcohols have been found in cleaning agents (ATSDR, 2018a). Washburn *et al*. (2005) reported perfluorooctanoate (PFO) concentrations ranging from <1 to 150 mg/L in product formulations of carpet-care solution, sealants, waxes, wax removers, paints, and cleaners.

5.2.2.5 Personal Care Products

A variety of PFAS have been used in personal care products such as dental floss, cosmetics, sunscreen, and shampoos (ATSDR, 2018a; US EPA, 2016a). PFOA and other PFAS have been used in hair conditioners to improve lubricity (*i.e.*, smoothness) (Kissa, 2001).

5.2.2.6 Apparel and Textiles

Clothing apparel and textiles, including carpets and upholstery, have been treated with a number of different PFAS because of their stain-resistant properties (Trudel *et al*., 2008). Fluoropolymers such as PTFE and non-polymer coatings have been used on clothing and outdoor gear, PFOA-based chromium has been applied to leather, and perfluoroalkyl acid (PFAA) and other PFAA precursors such as FTOHs and perfluoroalkyl sulfonamide alcohols have been sprayed on carpet to provide oil-, water-, and stain-resistant finishes (ITRC, 2020). PFOA and PFOS have also been detected in outdoor textiles, gloves, and awning cloths, while perfluorocarboxylic acids ranging from C5 (perfluoropentanoic acid) to C12 (perfluorododecanoic acid) were detected in articles of clothing (ATSDR, 2018a). PFOA concentrations in the formulations in treated apparel and home textiles ranged from <1 to 40 mg/L (<1,000,000 to 40,000,000 ng/L) (ATSDR, 2018a).

5.2.2.7 Other Miscellaneous Products

A wide variety of different PFAS have been used in other miscellaneous items such as batteries (Kissa, 2001), automobile interiors (US EPA, 2016a,b), car waxes (ITRC, 2020), and ski wax (Gomis *et al*., 2016; Fang *et al*., 2020). PFAS are applied to batteries to prevent the formation of dendrites (*i.e*., metallic microstructures that may impede battery performance) and to glass to aid in antifogging (Kissa, 2001). PFAS are also used in automobile waxes for their oil-, water-, and stain-resistant properties (ITRC, 2020). PFAS have been used in ski wax to reduce friction and repel dirt in order to enhance the performance of the skis (Buck *et al*., 2011). PFAS composition in ski wax varies by manufacturer, with some formulations including PFOA, PFCAs of varying carbon chain lengths up to 25 carbons, or fluorotelomer substances such as semifluorinated n-alkanes (SFAs) and alkenes (SFAenes) (Gomis *et al*., 2016; Fang *et al*., 2020; Buck *et al*., 2011). Fang *et al*. (2020) tested 11 different ski waxes for 26 PFAS and found C5-C10, C12-C14, and C16-C17 PFAS in all samples and PFBS, PFHxS, and PFOS in a subset of samples. In addition, total PFAS concentrations differed based on the form of ski wax, with higher concentrations in the ski wax powders (3,175-217,758 ng/g) than the ski wax blocks (265-7,660 ng/g) (Fang *et al*., 2020).

5.2.3 Industry

Multiple different PFAS (PTFE, FEP, PFA, PFOA, PFNA, PFHxA, and perfluoroether carboxylic acids [PFECAs]) have been manufactured in PFAS production facilities. For example, PFOA and ammonium perfluorooctanoate (APFO) were used in the manufacturing of PTFE. Since the voluntary phase-out of PFOA and PFOS, replacement PFAS are currently used in the production of a wide variety of materials (ITRC, 2020). PFAS use is also pervasive in a number of industries including aviation and aerospace, automotive, biocides (herbicides and pesticides), building and construction, cable and wiring, cosmetic and personal care products, electronics, energy, firefighting and safety, food processing, household products, medical products, metal plating, oil production, mining, paper and packaging, photolithography, semiconductor, and textiles (ITRC, 2020). PFAS composition varies depending on the industry. For example, PTFE, PFA, and PFSA salts are commonly used in the aviation industry, while PTFE is used in the electronics industry (ITRC, 2020). Thus, occupational exposures to different types and concentrations of PFAS in the manufacturing industry will vary among individuals depending on the industry in which they work.

5.2.4 Aqueous Film Forming Foams

Class B firefighting foams are found in chemical manufacturing plants, petroleum refineries, fire departments, vessels, off-shore drilling platforms, bulk storage facilities, airports, and military installations (US EPA, 2016a; ITRC, 2020). These foams are divided into two classes: fluorinated foams that contain PFAS and fluorine-free foams (F3) that do not contain PFAS. There are six different types of fluorinated foams that contain PFAS: (1) AFFF; (2) alcohol-resistant aqueous film-forming foam (AR-AFFF); (3) film-forming fluoroprotein foam (FFFP); (4) alcohol-resistant film-forming fluoroprotein foam (AR-FFFP); (5) fluoroprotein foam (FP); and alcohol-resistant fluoroprotein foam (FPAR) (ITRC, 2020). PFAS composition has been variable in AFFF over time, based on the type of foam and the production process used to make it (ITRC, 2020).

5.3 Transport and Exposure Media

PFAS can be released into the environment from the various identified point sources in Section 5.2, and the transport and distribution of the individual PFAS compounds are influenced by their physical-chemical

properties. PFAS released directly into the environment may be available for contact by an individual. PFAS concentrations in various exposure media are variable and depend on the PFAS source and magnitude of release; the fate and transport characteristics of released PFAS, which are dependent on the chemical characteristics of the individual PFAS (*e.g.*, solubility, mobility, persistence, bioaccumulation) and the exposure medium. Primary environmental exposure media include surface water, drinking water, sediment, soil, air, and house dust. Secondary exposure media include dietary items such as produce, livestock or game, fish or shellfish, and inedible items that are incidentally consumed. Exposure to these media can occur *via* ingestion, dermal contact, and/or inhalation. Given the wide variety of transport mechanisms and exposure media, exposures to specific PFAS can vary greatly between individuals, depending on the types of contact the individuals have with these media that may be impacted from a variety of sources. Tracing detected blood levels of particular PFAS in individuals to their ultimate sources would require an individualized exposure assessment.

5.3.1 PFAS from Waste Materials

PFAS in waste materials are released into the environment *via* landfill leachate, effluents, biosolids, and stack emissions. PFAS in waste materials may be released into soil *via* landfill leachate or through the application of biosolids. Although modern landfills are equipped with leachate collection systems, elevated concentrations of PFOS have been detected in groundwater wells near landfills due to physical compromises or biological reactions under anaerobic conditions within the landfill (US EPA, 2016b; ITRC, 2002). Multiple studies have detected PFOA and PFOS concentrations ranging from 55 to 2,531 and 589 to 1,409 ng/g, respectively, in surface soil samples where municipal biosolids were applied (Brusseau *et al.*, 2020; ATSDR, 2013). These soils may be a PFAS source for surface water, groundwater, the atmosphere, and biota.

Certain PFAS are mobile in soil by rainfall and can migrate to underlying groundwater aquifers and/or nearby surface water bodies used for drinking water sources (ATSDR, 2018a; ITRC, 2020; Clarke and Smith, 2011; US EPA, 2016a,b). PFAS migration through soil is dependent on a variety of factors, including source (*e.g.*, type of PFAS, co-contaminants), meteorological conditions, and soil properties (Brusseau *et al.*, 2020). Studies have found varying levels of transport through soils depending on the specific PFAS (Lindstrom *et al.*, 2011a).

In addition, studies have found uptake of certain PFAS from biosolids application to crops, subsequently consumed by consumers, livestock, and farm animals (Sunderland *et al.*, 2019; ATSDR, 2018a; US EPA, 2016a,b; Vestergren and Cousins, 2009).

WWTP effluent is considered a major source of PFAS to receiving surface water bodies because conventional sewage treatment methods do not effectively remove certain PFAS (ITRC, 2020). In addition, multiple studies found higher concentrations of some PFAS (*e.g.*, PFNA, PFOA, PFOS, PFOSA) in the effluent than the influent, suggesting that the waste treatment process may promote the biodegradation of some precursors contributing to the increase in concentration of certain PFAS in the effluent (Loganathan *et al.*, 2007; Schultz *et al.*, 2006; Sinclair and Kannan, 2006; Hu *et al.*, 2016). Due to their low volatility and high chemical stability, PFOA and PFOS have been detected in multiple rivers and lakes that serve as drinking water sources or recreational areas throughout the continental US at concentrations ranging from <1 to 1,150 ng/L for PFOA and <1 to 319 ng/L for PFOS (US EPA, 2016a,b).

Some PFAS in surface water may bioaccumulate (*i.e.*, uptake by an organism *via* exposure to water and/or diet) in fish and other aquatic organisms, resulting in potential dietary exposures for human consumers (US EPA, 2016a,b). PFOS has been detected in fish collected near the outfall of a WWTP, with tissue concentrations up to 400 ng/g (ITRC, 2020). Bioaccumulation studies of the food chain have found that

certain long-chain PFAS bioaccumulate more readily than short-chain PFAS and certain sulfonates are retained more readily than carboxylates (Lindstrom *et al*., 2011b). Long-chain PFAS, such as PFOA and PFOS, are commonly found in fish collected from areas of known point sources (Fromme *et al*., 2009) and fish consumption could be a source of exposure to certain PFAS for some individuals (Domingo and Nadal, 2017; Fromme *et al*., 2009).

PFAS in surface waters may lead to secondary and tertiary exposure media. For example, surface water can be used to irrigate produce that is consumed by humans and/or livestock, and the livestock can be subsequently consumed by humans. Studies have found uptake of certain PFAS into lettuce and strawberries irrigated with waters containing PFAS (Domingo and Nadal, 2017). Similarly, elevated PFOA and PFOS concentrations were observed in soils where contaminated surface water was used for irrigation (Brusseau *et al*., 2020).

Landfills, WWTPs, and certain types of incinerators may also be a point source of PFAS for outdoor air. Elevated PFAS concentrations were found in air samples collected in areas near landfills, WWTPs, and incinerators (ITRC, 2020). Ambient air samples near landfills and WWTPs detected PFAS concentrations 1.5-15 times higher than background reference samples (ITRC, 2020).

5.3.2 PFAS from Consumer Products

Individuals may be exposed to PFAS in consumer products through multiple exposure scenarios. Consumer products may be incidentally ingested. For example, PFAS from food packing materials may leach into foods that are subsequently consumed (Begley *et al*., 2005; Guo *et al*., 2019) or PTFE coatings on non-stick cookware may be dislodged and incidentally ingested (Sajid and Ilyas, 2017). Consumers may also incidentally ingest PFAS from hand-to-mouth contact when touching a surface treated with PFAS and then mouthing their hand. Washburn *et al*. (2005) tested the amount of extractable polyfluorooctanoate (PFO) in treated carpets, upholstery, apparel, and textiles using water, saliva, and perspiration to simulate human exposures and found that the amount available for exposure is a fraction of the concentration of the final product.

PFAS in consumer products may also be dermally absorbed, although the dermally absorbed fraction of PFAS is generally considered to be low (ATSDR, 2018a; ITRC, 2020). Peaslee *et al*. (2020) hypothesized that firefighters may be exposed to PFAS from direct contact with the skin to PFAS-treated personal protective equipment but noted that further studies are needed to deduce the fraction of PFAS in the blood that is attributable to dermal absorption. Consumer products such as ski wax may release PFAS into the environment from product use, as evident in the presence of PFAS detected in snowmelt, surface water, and soils near ski resorts (ITRC, 2020; Buck *et al*., 2011). Consumer products may also produce particulates or gases that may be inhaled by an individual. For example, physical degradation of PFAS-treated textiles, upholstery, and carpets can be a source of PFAS in house dust available for inhalation (ITRC, 2020; ATSDR, 2018a; Trudel *et al*., 2008).

5.3.3 PFAS from Industry

PFAS from industry may be released into the environment *via* accidental releases (*e.g*., spills and leaks), permitted releases, and stack and fugitive air emissions. PFAS have been detected in air, water, and soil surrounding manufacturing sites that use and manufacture PFAS. PFOA and PFOS concentrations up to 470 ng/g and 189 ng/g, respectively, were measured in soil samples collected near PFAS manufacturing facilities (Brusseau *et al*., 2020; US EPA, 2016a,b). Soil samples near a PFAS manufacturing facility found higher PFOA and PFOS concentrations at deeper depths, signifying migration through soils (US EPA, 2016a,b). Groundwater samples collected near a PFAS manufacturing facility reported PFOA,

perfluorobutanoic acid (PFBA), PFOS, PFHxS, and perfluorobutane sulfonic acid (PFBuS) concentrations of 24.6-619 µg/L, 23.3-318 µg/L, 26 µg/L, 6.47-40 µg/L, and 2.11-26.1 µg/L, respectively (ATSDR, 2013). Groundwater neighboring PFAS industrial sites can be used as a drinking water source or be hydraulically connected to drinking water sources for consumption.

Ambient air concentrations near PFAS manufacturing facilities reported PFOA concentrations up to 1 $\mu g/m^3$, suggesting that inhalation may be a source of exposure for workers at the plant (Vestergren and Cousins, 2009). Elevated PFAS particulate levels have been detected in air samples collected along the fence line of a PFAS manufacturing facility in West Virginia, which could result in exposure to nearby communities (ATSDR, 2018a). Elevated concentrations of polyfluorinated sulfomide (N-methyl perfluorooctanesulfonamidoethanol, N-ethyl perfluorooctanesulfonamidoethanol, and N-EtFOSA) were detected in air samples collected in the vicinity of a carpet processing factory (Fromme *et al*., 2009). Data have suggested that PFAS emitted into the air during manufacturing could be transported by wind to nearby well fields and deposited onto surface soils and subsequently migrate to the underlying aquifer (Lau *et al*., 2007; ATSDR, 2018a).

5.3.4 PFAS from AFFF

PFAS in AFFF may be released into the environment at varying intensities depending on the situation. Low volumes of release may occur during storage and transfer, while more significant volumes of release may occur during apparatus testing, firefighting, or fire-training episodes (ITRC, 2020). Surface water and groundwater near areas where foams have been used may contain residual PFAS. PFAS attributed to AFFF have been detected in groundwater near fire-training areas in Michigan at concentrations ranging from 4 to 120 µg/L (or 4,000-120,000 ng/L) (ATSDR, 2018a). PFOS and PFOA have been detected at concentrations up to 8,790 and 3,750 ng/L, respectively, in surface waters near areas where AFFF was applied (ITRC, 2020). Brusseau *et al*. (2020) reported a wide range of concentrations of 10 different PFAS in surface soils collected from US military installations where AFFF was used (PFBA: 0.1-820 ng/g; PFHxA: 0.07-15,300 ng/g; PFOA: 0.07-50,000 ng/g; PFDA: 0.03-430 ng/g; PFBS: 0.05-5,550 ng/g; PFHxS: 0.07-21,000 ng/g; PFOS: 0.09-373,000 ng/g; PFDS: 0.05-640 ng/g; PFOSA: 0.09-20,000 ng/g; and 6:2 FTSA: 0.2-68,000 ng/g). Anderson *et al*. (2016) collected surface soil, subsurface soil, sediment, surface water, and groundwater samples from 40 sites at 10 military installations throughout the US where AFFF was used between 1970 and 1990 and analyzed 16 different PFAS (PFBA, PFBS, PFPA, PFHxA, PFHxS, PFHpA, PFOA, PFOSA, PFOS, PFNA, PFDA, PFDS, PFUnA, PFDoA, PFTriA, PFTeA). All 16 PFAS were detected in all media except for PFTriA and PFTeA, which were not detected in any surface water samples. PFOS had the highest detection frequency in soil and sediment samples while PFHxA and PFHxS had the highest detection frequencies in surface water and groundwater samples, respectively. While PFAS concentrations varied widely by media, PFOS concentrations were the highest in all media, with a wide range of concentrations in each specific type of media (surface soil: <LOQ - 9,700 ng/g; subsurface soil: <LOQ - 1,700 ng/g; sediment: <LOQ - 190,000 ng/g; surface water: <LOQ - 8,970,000 ng/L; groundwater:<LOQ - 4,300,000 ng/L). Similarly, FTSA has been detected in groundwater, soil, and biota samples collected at military installations, firefighting sites, and areas where AFFF has been used (Buck *et al*., 2011; ATSDR, 2018a).

5.3.5 PFAS from Other Sources

Multiple studies have detected PFAS in various environmental media that have not been attributed to an identified point source, making it difficult to reduce exposures from these sources. For example, elevated PFAS concentrations have been detected in the US drinking water supply that are not associated with known PFAS sources (Sunderland *et al*., 2019 ; Hu *et al*., 2016). PFAS from street runoff or stormwater runoff from nonpoint sources entering a receiving surface water body may be contributing sources (ITRC, 2020).

PFAS not associated with a point source have also been detected in ambient air worldwide. Samples collected in an urban area of Albany, NY, detected multiple PFSA such as PFOS and PFOA in the gas and particulate phase (PFOS: 0.4-1.2 pg/m^3 as a particulate and 0.9-3.0 pg/m^3 as a gas; PFOA: 0.8-4.2 pg/m^3 as a particulate and 1.9-6.5 pg/m^3 as a gas) (ATSDR, 2018a; Fromme *et al*., 2009).

PFAS have also been detected in an array of food items that are harvested in areas of no known point sources. A market basket study sampled fish sold in fish markets and grocery stores for consumption and detected several different long-chain PFAS, including PFOS and PFOA. Many of these samples were collected in commercial fishing areas associated with no known point sources (Ruffle *et al*., 2020). Similarly, PFOS was detected in milk and ground beef samples and PFOA was detected in green beans, apples, bread, and ground beef sold in markets within the US at concentrations up to 0.85 ng/g fresh weight for PFOS and 2.35 ng/g for PFOA (Fromme *et al*., 2009).

PFAS in the environment can be the result of a breakdown of a precursor compound. For example, large molecules such as DiPAPs are found in food packaging, and WWTP sludge releases fluorotelomer alcohol (FTOH) that may degrade into PFOA. Various studies have found PFAS in remote locations worldwide, where the sources are unknown. Long-range transports (*via* air and ocean currents) and transformations (*e.g*., atmospheric photooxidation, biotransformation, dissociation under acidic conditions) of precursors such as fluorotelomer alcohols and perfluoroalkyl sulfonamides have resulted in PFOA and PFOS contamination in remote locations with no known point source (ATSDR, 2018a; US EPA, 2016a,b). For example, soil samples collected in Antarctica detected PFOA and PFOS at 0.048 and 0.007 ng/g, respectively (ITRC, 2020).

5.4 Receptors

A receptor is an exposed individual or population. Receptors can be those within the general population (adult, child, infant) or workers exposed to PFAS from the various exposure pathways discussed in Section 5.3. PFAS exposure and magnitude of intake are variable and largely dependent on the receptor and the rate at which the particular PFAS at issue enters the body. As discussed in Section 5.1, PFAS can potentially enter the body from three exposure pathways (*i.e*., ingestion, dermal contact, and inhalation), although the dermal route is generally thought to be a limited pathway (ITRC, 2020).

Many PFAS exposures to the general population are from the ingestion pathway (*e.g*., consumption of food contaminated with PFAS from food packaging materials, food grown in areas where biosolids were applied, hand-to-mouth transfer of treated consumer products, food cooked on PTFE-coated cookware, seafood found in surface waters impacted by PFAS, mouthing by infants) (Washburn *et al*., 2005). Inhalation (*e.g*., of house dust or aerosols from impregnation sprays, or of air emissions downgradient of a facility's stack and fugitive emissions) and dermal exposures (*e.g*., to liquids and treated apparel) to PFAS are present, but to a lesser degree than ingestion (ITRC, 2020). The median uptake of PFOS and PFOA for the general population was estimated to be 2 and 3 ng/kg-day, respectively, from non-occupational exposures *via* indoor and outdoor air, house dust, drinking water, and diet, with diet contributing 90% of the exposure (Lindstrom *et al*., 2011b). Within the general population, a subset of consumers are individuals who are exposed to certain products (*e.g*., people who use personal care products containing PFAS) or dietary items (*e.g*., fish and produce consumers).

Occupational exposure includes workers' exposure from PFAS manufacturing or manufacturing processes involving PFAS, as well as workers' exposure to PFAS-containing products (*e.g*., carpet installers, firefighters, upholsterers, waste handlers). Occupational exposures occur primarily through inhalation, although incidental ingestion and dermal contact may contribute (Vestergren and Cousins, 2009; ATSDR, 2018a). Workers who are exposed to PFAS in their occupations are likely to also have non-occupational

sources of exposure as well. While some occupationally exposed workers have higher serum levels of PFAS than the general public (as discussed below in Section 6.2), the levels of body burden have decreased over time, which may be related to the phase-out of certain PFAS production, improved working conditions, lower emissions from processes, or a combination of these factors (Fromme *et al*., 2009; Vestergren and Cousins, 2009). Moreover, occupational exposures to PFAS are variable, depending on the type of job. Individuals working directly with PFAS, such as in manufacturing processes, would be expected to have higher PFAS exposure than those with more limited contact, such as carpet installers or other types of workers who handle PFAS-containing products (ATSDR, 2018a).

5.4.1 Intake

Exposure and susceptibility to exposure are not uniform across the population because an individual's intake varies based on the magnitude, frequency, duration, and route of exposure. For example, exposure or magnitude of intake of PFAS *via* drinking water consumption varies based on the detected concentration, intake rates, residence factors, and behavioral factors. I discuss each of these factors below to emphasize the variability for the drinking water exposure pathway as an example, because it is a common exposure pathway for PFAS, but the intake factors will also vary for other PFAS exposure pathways besides drinking water ingestion. US EPA's "Exposure Factors Handbook" provides many examples of variability in other intake factors among individuals, including inhalation rates, frequency of hand contact with objects or surfaces, hand-to-mouth contact frequency, and ingestion rates of certain types of foods (US EPA, 2011b).

Drinking water concentrations are highly variable; for example, Vestergren and Cousins (2009) found PFOA concentrations ranging from <0.32 to 7,200 ng/L (Vestergren and Cousins, 2009). Similarly, Hu *et al*. (2016) found concentrations as high as 349 ng/L for PFOA, 1,800 ng/L for PFOS, and 56 ng/L for PFNA in drinking water samples from 66 public water supplies. As noted earlier, PFAS concentrations in drinking water near military installations where AFFF was used had a large range of concentrations. Depending on which source of drinking water is consumed and the rate of consumption, the intake by an individual may differ.

US EPA (2019d) has compiled drinking water ingestion rates based on values reported in multiple studies. As presented in Table 5.1, US EPA's analysis of the 2005-2010 National Health and Nutrition Examination Survey (NHANES) data found varying direct (water ingestion as a beverage) and indirect (water added in preparation to food or beverage) drinking water ingestion rates by age group. Because of differences in intake rates and in body weight, there is even considerable variability in body weight-normalized drinking water intakes within the same age group (*e.g*., 21-30 year olds with 5th and 95th percentile drinking water ingestion rates of 0.7 and 46.9 mL/kg-day, respectively, which is a 67-fold difference; 1-2 year olds with 5th and 95th percentile drinking water ingestion rates of 1.4 and 57.3 mL/kg-day, respectively, which is a 41-fold difference). The drinking water ingestion rates in Table 5.1 are only for consumers of community water supplies and do not account for the population in the survey that did not ingest drinking water from those sources during the two-day survey period.

Table 5.1 Drinking Water Ingestion Rates

Age Group	Sample Size	5th Percentile	50th Percentile	95th Percentile
Birth to <1 month	20	4.4	151.9	224.0
1 to <3 months	45	26.4	134.0	267.2
3 to <6 months	65	12.0	91.0	158.4
6 to <12 months	244	4.4	63.9	133.0
1 to <2 years	394	1.4	18.5	57.3
2 to <3 years	445	3.1	17.5	66.6
3 to <6 years	860	1.7	14.4	45.2
6 to <11 years	1,473	1.2	11.5	40.8
11 to <16 years	1,449	0.5	6.8	31.1
16 to <21 years	1,312	0.3	6.2	31.1
21 to <30 years	1,318	0.7	11.7	46.9
30 to <40 years	1,530	1.4	12.4	43.6
40 to <50 years	1,532	1.6	13.5	43.3
50 to <60 years	1,412	1.8	14.9	41.8
60 to <70 years	1,453	2.5	14.8	39.8
70 to <80 years	1,017	3.7	14.2	37.0
80+ years	650	3.6	14.1	32.7

Note: Drinking water ingestion rates are two-day averages of drinking water consumption from community water supplies and are reported in mL/kg-day.
Source: US EPA (2019d).

Drinking water ingestion rates can also vary by the water supply associated with a given residence. For example, US EPA's drinking water ingestion rates presented in Table 5.1 are for residences connected to community water supplies, but they do not account for residences connected to alternate sources of drinking water (*e.g.*, private wells, bottled water, springs). In addition, an individual's exposure from drinking water intake may depend on where the water is collected and the public water supply associated with that location. For example, an individual may be exposed to multiple sources of water throughout the day depending on where they spend their time. School-aged children may spend, on average, six hours a day for nine months out of the year at school (US EPA, 2011b) and could be exposed to PFAS from a drinking water source that is different from the one connected to their residence. In addition, a resident's exposure to a specific water supply is dependent on their exposure frequency (number of days a year at the residence drinking water) and exposure period (number of years at the residence drinking water). Table 5.2 presents the variability in residential occupancy reported by US EPA (2011b). Given the variability of residential occupancy, residents move at least 1-2 times in their lifetime (assuming US EPA's recommended life expectancy of 78 years for men and women [US EPA, 2011b]) and may be exposed to different water supplies and/or other potential sources of PFAS at each residence.

Table 5.2 Residential Occupancy Period

Group	Sample Size	5th Percentile	50th Percentile	95th Percentile
Males and Females	500,000	2	9	33
Males Only	244,274	2	8	31
Females Only	255,726	2	9	35

Note: Residential occupancy period reported in years.
Source: US EPA (2011b).

Drinking water ingestion rates can also vary based on other factors such as life stage (US EPA [2019d] reported variable water ingestion rates among pregnant, lactating, and child-bearing age women), climate (hot *versus* cold climates), and activity (McNall and Schlegel [1968, as cited in US EPA, 2019d], found variable water ingestion rates based on the activity level of an individual). Consequently, the intake for a

drinking water scenario for residents may vary vastly depending on the scenario and individual. Given that drinking water may be a portion of an individual's daily PFAS exposure, when accounting for other exposure pathways for a given receptor, the variability grows with each additional exposure pathway.

Similarly, a firefighter's PFAS exposure from AFFF may vary depending on a multitude of factors related to the foam (*e.g.*, age and type of foam, PFAS formulation in AFFF), the use (*e.g.*, frequency of use, exposure duration, magnitude of release), and the individual (*e.g.*, use of personal protective equipment to minimize exposures, age, race, body mass, occupational tenure). Multiple studies have found higher PFAS serum concentrations among firefighters than the general population (as discussed below in Section 6.3) but could not determine if the higher concentrations were due to exposures to AFFF, PFAS exposure from firefighter gear, or consumption of PFAS-contaminated drinking water near areas where AFFF was used (ITRC, 2020; ATSDR, 2018a).

Given the wide variability of intake between individuals, even among those who have contact with the same exposure media impacted by the same PFAS source and type, individual assessment is necessary to reliably estimate exposures for any particular individual.

5.4.2 Risk

Exposure to or intake of a chemical does not indicate that risk or harm has occurred. Risk is the chance of harmful effects from exposure to a chemical that can induce an adverse response. Risk involves comparing an individual's PFAS dose (amount of PFAS intake that is available for interaction with target tissues) to levels known to induce an adverse response (US EPA, 2005). Individual variability factors such as intake, body mass, age, gender, pregnancy status, and (in the case of certain PFAS) menstruation are some of the critical determinants of chemical dose and risk. Individual variability can be grouped as intra- and interindividual variability. Intra-individual variability occurs from changes in an individual over time, such as physical (*e.g.*, body weight and age) and behavioral changes (*e.g.*, ingestion rates) (US EPA, 2019a). For example, lactating women are likely to ingest higher concentrations of PFAS from drinking water than non-lactating women due to their increased water intake rate needed to aid milk production (US EPA, 2016a,b). Interindividual variability, or differences among individuals within a population, can also impact PFAS intakes. For example, while a community may be connected to a public water supply containing a certain concentration of PFAS, individual doses within the community will differ based on specific factors (*e.g.*, age, gender, occupation, diet, race, behavior, intake rate). Variability in these factors will result in variability in exposure, dose, and risk; thus, the evaluation of potential health risks (if any) from exposure to PFAS requires assessment of intakes on an individualized basis. Further, as demonstrated above, given the wide variety of potential sources of PFAS exposures, the wide variety of individual PFAS with different properties, and the varying pathways of potential exposures, tracing any such risk to one or more individual sources generally requires an extensive and individualized exposure assessment.

6 Variability in Serum Concentrations

In this section, I describe the variability in serum concentrations of PFAS in the general population, worker populations with known exposures to PFAS, and firefighters. I focus on serum concentrations of PFOA, PFOS, PFHxA, and PFNA as examples because these PFAS are well studied with respect to serum concentrations across populations compared to other PFAS. I focus on describing the serum concentrations of these PFAS as measured in different populations within the last 10 years, if available, as they are most representative of today's population. The variability in serum concentrations reported in the studies in this section indicates that PFAS exposures will vary significantly among proposed class members. Moreover, there is variability among individuals in the sources of PFAS that contribute to the variability in exposures and serum concentrations. Thus, the evaluation of potential health effects (if any) from those exposures requires individualized assessment.

6.1 General Population

Concentrations of certain PFAS have been measured in the serum of individuals in the general population, including NHANES participants, American Red Cross blood donors, and a population living in New York City. Table 6.1 summarizes the range, 50th percentile (median), and 95th percentile serum concentrations of PFOA, PFOS, PFHxS, and PFNA, as measured most recently in these populations. The serum concentrations of each of the PFAS in Table 6.1 are highly variable both within and among the different populations. For example, the range of serum concentrations across the studies varies from below the limit of detection to 13.3 ng/mL for PFOA and from 0.2 to 29.4 ng/mL for PFOS. Median concentrations of each of the PFAS are slightly higher in NHANES participants compared to American Red Cross blood donors and the population in New York City.

Table 6.1 Serum PFAS (ng/mL) in Community Populations in the United States

Cohort/Population	N	Range (Min-Max)	50th Percentile (95% CI)	95th Percentile (95% CI)	Reference
PFOA					
NHANES (2015-2016)	1,993	NR	1.57 (1.47-1.77)	4.17 (3.87-4.67)	CDC (2019)
American Red Cross Donors (2015)	616	LOD – 13.3	1.1	3.2	Olsen *et al*. (2017)
World Trade Center Health Registry Comparison Population (2014-2016)[a]	185	NR	1.39 (IQR: 0.75)	NR	Trasande *et al*. (2017)
PFOS					
NHANES (2015-2016)	1,993	NR	4.80 (4.40-5.30)	18.3 (15.5-22.7)	CDC (2019)
American Red Cross Donors (2015)	616	0.2-29.4	4.3	8.6	Olsen *et al*. (2017)
World Trade Center Health Registry Comparison Population (2014-2016)[a]	185	NR	2.78 (IQR: 2.18)	NR	Trasande *et al*. (2017)
PFHxS					
NHANES (2015-2016)	1,993	NR	1.20 (1.10-1.40)	4.90 (4.10-5.80)	CDC (2019)
American Red Cross Donors (2015)	616	LOD – 42.2	0.9	3.5	Olsen *et al*. (2017)
World Trade Center Health Registry Comparison Population (2014-2016)[a]	185	NR	0.53 (IQR: 0.47)	NR	Trasande *et al*. (2017)
PFNA					
NHANES (2015-2016)	1,993	NR	0.6 (0.5-0.6)	1.90 (1.50-2.20)	CDC (2019)
American Red Cross Donors (2015)	616	LOD-4.0	0.4	1.1	Olsen *et al*. (2017)
World Trade Center Health Registry Comparison Population (2014-2016)[a]	185	NR	0.49 (IQR: 0.33)	NR	Trasande *et al*. (2017)

Notes:
CI = Confidence Interval; IQR = Interquartile Range (25th to 75th percentile); LOD = Limit of Detection; N = Number of participants/samples; NHANES = National Health and Nutrition Examination Survey; NR = Not Reported; PFAS = Perfluoroalkyl Substances; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctanesulfonic Acid; PFNA = Perfluorononanoic Acid; PFHxS = Perfluorohexane Sulfonate
(a) Participants were born between September 11, 1993, and September 11, 2001, did not reside or attend school south of Canal St. in New York City, NY, and were not present south of Canal St. on the morning of September 11, 2001.

Table 6.2 summarizes the median and 95th percentile serum concentrations of PFOA, PFOS, PFHxS, and PFNA by sex in the most recently reported NHANES cycle (2015-2016). For each of these PFAS, the serum concentrations are lower in females than males, on the order of 1.8-fold lower for PFOS and PFHxA and 1.2- to 1.3-fold lower for PFHxA and PFNA. Lower serum concentrations of these PFAS were consistently observed in females in all of the earlier NHANES cycles going back to 1999-2000 (CDC, 2015, 2019), as well as in American Red Cross donors with serum measurements of PFAS in 2000-2001, 2006, 2010, and 2015 (Olsen *et al*., 2017). Serum PFAS concentrations also vary among women based on their pregnancy status. Although more recent data are not available, Woodruff *et al*. (2011) reported lower serum concentrations of PFOA and PFOS in pregnant women compared to non-pregnant women in the 2003-2004 NHANES cycle. For example, the median serum PFOA concentration was 23% higher in non-pregnant

women (3.2 ng/mL) compared to pregnant women (2.6 ng/mL). Reasons for the sex difference and the difference based on pregnancy status are discussed below in Section 7.1.4

Table 6.2 Serum PFAS (ng/mL) in NHANES 2015-2016 Cohort by Sex

Sex	N	50th Percentile (95% CI)	95th Percentile (95% CI)
PFOA			
Male	964	1.87 (1.67-2.07)	4.07 (3.67-4.87)
Female	1,029	1.37 (1.27-1.47)	4.17 (3.67-4.97)
PFOS			
Male	964	6.40 (5.60-6.70)	21.3 (17.4-24.4)
Female	1,029	3.60 (3.30-3.90)	14.6 (12.2-18.3)
PFHxS			
Male	964	1.60 (1.50-1.70)	5.80 (4.40-6.40)
Female	1,029	0.90 (0.80-1.00)	3.80 (2.90-5.70)
PFNA			
Male	964	0.60 (0.60-0.70)	1.90 (1.60-2.20)
Female	1,029	0.50 (0.50-0.60)	1.80 (1.40-2.50)

Notes:
CI = Confidence Interval; N = Number of participants/samples; NHANES = National Health and Nutrition Examination Survey; PFAS = Perfluoroalkyl Substances; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctanesulfonic Acid; PFNA = Perfluorononanoic Acid; PFHxS = Perfluorohexane Sulfonate.
Source: CDC (2019).

Table 6.3 summarizes serum concentrations of PFOA, PFOS, PFHxS, and PFNA by age, as measured in the 2015-2016 NHANES cycle. The serum concentrations of each of these PFAS were higher in adults than in teenagers in this cohort, but trends by age have not been consistently reported across studies. Some found no clear trend in concentration by age (Frisbee *et al*., 2009; Vestergren and Cousins, 2009; Olsen *et al*., 2017; Jian *et al*., 2018), whereas others found associations with age that, in some cases, differed by sex. For example, an analysis of data from four NHANES cycles (1999-2008) showed that while serum PFOS concentrations increased with age in males and females, serum PFOA concentrations increased as age increased in females and declined as age increased in males (Kato *et al*., 2011). These differing trends may be attributable to sex-related differences in dose based on physiology, as discussed below in Section 7.1.4. Some of the differences in age groups may also be influenced by the patterns of PFOA and PFOS usage over time; older individuals likely had higher exposures to PFOA and PFOS in products than children born after the PFOA and PFOS phase-out, which began in 2002 (3M, 2020).

Table 6.3 Serum PFAS (ng/mL) in NHANES 2015-2016 Cohort by Age

Age (years)	N	50th Percentile (95% CI)	95th Percentile (95% CI)
PFOA			
12-19	353	1.27 (1.17-1.47)	2.47 (2.07-2.97)
≥ 20	1,640	1.67 (1.57-1.87)	4.27 (4.07-4.97)
PFOS			
12-19	353	2.90 (2.70-3.30)	6.60 (6.10-7.70)
≥ 20	1,640	5.20 (4.80-5.70)	19.1 (15.8-24.4)
PFHxS			
12-19	353	0.90 (0.70-1.20)	3.10 (2.30-5.80)
≥ 20	1,640	1.30 (1.20-1.40)	5.00 (4.10-6.20)
PFNA			
12-19	353	0.50 (0.40-0.60)	1.20 (1.00-1.30)
≥ 20	1,640	0.60 (0.50-0.60)	1.90 (1.60-2.30)

Notes:
CI = Confidence Interval; N = Number of participants/samples; NHANES = National Health and Nutrition Examination Survey; PFAS = Perfluoroalkyl Substances; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctanesulfonate; PFNA = Perfluorononanoic Acid; PFHxS = Perfluorohexane Sulfonate.
Source: CDC (2019).

Several studies have reported that PFAS serum concentrations can vary by race. For example, Mexican-Americans had lower serum concentrations of PFOA, PFOS, PFHxS, and PFNA than non-Hispanic whites or non-Hispanic blacks in the NHANES cycles encompassing 2011-2016 (CDC, 2019). Asians had higher serum concentrations of PFOS and PFNA than blacks, whites, and Hispanics in the 2011-2016 NHANES cycles (CDC, 2019). Other studies report that serum concentrations of PFAS appear to be associated with dietary habits, lifestyle factors, and income level (Jian *et al*., 2018; Sunderland *et al*., 2019).

6.2 Workers Exposed to PFAS

In addition to general population exposure sources, workers may be exposed to PFAS during the production of PFAS or the manufacturing or occupational use of products containing PFAS. Worker exposure monitoring through measurement of serum PFAS levels has occurred at various facilities where PFAS are present. In general, the serum concentrations of PFAS measured in these working populations are associated with exposures to PFAS that are typically higher than exposures that would be expected in the general population and communities with point sources of exposure. Exposure monitoring for PFAS was conducted at 3M and DuPont PFAS production facilities in particular, and below, I present data on serum concentrations of PFAS in these workers to illustrate variability among worker populations at different facilities and even within the populations at specific facilities. There are other worker populations involved in the production of PFAS or the manufacture or use of products containing PFAS that I did not evaluate here, and their exposures would be expected to differ, particularly based on the types and amounts of PFAS present. I discuss firefighters specifically in Section 6.3 below, however, as another worker population potentially exposed to PFAS. As the most recent data are limited for these populations, Table 6.4 summarizes the serum concentrations of PFOA and PFOS since 2000 in workers at 3M and DuPont facilities with exposure to PFAS. This table shows that serum concentrations of PFOA and PFOS vary substantially across the worker populations, even within the same facility.

Table 6.4 Serum PFAS (ng/mL) in Occupational Populations in the United States

Time Period/Population	Cohort Details	N	Range	Median	Reference
PFOA					
2000 (3M, Decatur, AL)		263	40 – 12,700	NR	Olsen *et al*. (2003a)
2000 (3M, Cottage Grove, MN)	Male workers only	122	10-92,030	950	Olsen and Zobel (2007)
2000 (3M, Decatur, AL)		188	40-12,700	1,510	
2002 (3M, Decatur, AL)	Males (41) and females (13)	54	25-4,810	NR	Olsen and Mandel (2003a)
2002 (3M, Cottage Grove, MN)		38	70-32,600	1,600	Olsen and Mandel (2003b)
2004 (DuPont Washington Works, WV)	Current PFOA exposure	259	17.4-9,550	494	Sakr *et al*. (2007)
	Intermittent current exposure	160	8.1-2,070	176	
	Past exposure	264	8.6-2,590	195	
	No exposure	342	4.6-963	114	
2005-2006 (DuPont Washington Works, WV)		1,881	NR	113	Steenland *et al*. (2015)
2005-2006 (C8 Health Project Cohort Dupont Worker)		3,713	55.9-256.2[a]	112.7	Winquist and Steenland (2014)
PFOS					
2000 (3M, Decatur, AL)	Male workers	188	60-4,170	1,000	Olsen and Zobel (2007)
2000 (3M, Cottage Grove, MN)		122	30-4,790	450	
2000 (3M, Decatur, AL)		263	60-10,060	NR	Olsen *et al*. (2003a)
2002 3M, Decatur, AL)	Males (41) and females (13)	54	82-4,580	NR	Olsen and Mandel (2003a)
2002 (3M, Cottage Grove, MN)		38	50-1,170	280	Olsen and Mandel (2003b)

Notes:
N = Number of participants/samples; ND = Non-detectable; NR = Not Reported; PFAS = Perfluoroalkyl Substances; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctanesulfonate.
(a) 25th to 75th percentile.

6.3 Firefighters

Few studies have measured serum concentrations of PFAS in populations of firefighters. Table 6.5 summarizes the serum concentrations of PFOA, PFOS, PFHxS, and PFNA measured in the available studies. The serum concentrations of each of these PFAS were variable across the different populations of firefighters studied. Median serum concentrations of each of the PFAS were higher in firefighters compared to those with other employment in the C8 Health Project in Ohio and West Virginia, though for PFOS, PFHxS, and PFNA, the maximum concentrations in the serum of non-firefighters were higher than in the serum of firefighters (Jin *et al*., 2011). Serum concentrations of PFOA, PFOS, and PFHxS were slightly higher in firefighters in California compared to the general population from the 2009-2010 NHANES cycle, whereas PFNA concentrations were slightly higher in the general population than in the California firefighters (Dobraca *et al*., 2015). A study of firefighters in Ohio in the NHANES 2018-2019 cycle shows variability in serum concentrations of PFOA, PFOS, and PFHxS between airport and suburban firefighters, though serum concentrations of PFNA do not differ between the two types of firefighters (Leary *et al*., 2019). As with other groups occupationally exposed to PFAS, firefighters' PFAS blood levels vary widely.

Table 6.5 Serum PFAS (ng/mL) in Firefighters

Cohort	Cohort Details	N	Median	Range		Reference
				Min	Max	
PFOA						
C8 Health Project (2005-2006), Ohio or West Virginia	Male firefighters (median employment 12.5 years)	36	31.5	0.25	7,534.6	Jin *et al*. (2011)
	Other employment	5,373	26.9	0.60	1,925.2	
FOX Study (2010-2011), California	Firefighters	101	3.86	NR	18.1	Dobraca *et al*. (2015)
	NHANES (2009-2010)	876	3.7	NR	24	
NHANES (2018-2019), Southwest Ohio	Airport firefighters	36	2.17	1.10	4.65	Leary *et al*. (2019)
	Suburban firefighters	9	1.72	1.02	3.07	
	Combined firefighters	45	2.15	1.02	4.65	
PFOS						
C8 Health Project (2005-2006), Ohio or West Virginia	Male firefighters (median employment 12.5 years)	36	27.85	0.25	67.5	Jin *et al*. (2011)
	Other employment	5,373	23.0	0.25	564.3	
FOX Study (2010-2011), California	Firefighters	101	12.7	NR	46.6	Dobraca *et al*. (2015)
	NHANES (2009-2010)	876	12.3	NR	281	
NHANES (2018-2019), Southwest Ohio	Airport firefighters	36	10.69	4.28	30.42	Leary *et al*. (2019)
	Suburban firefighters	9	4.04	1.57	9.34	
	Combined firefighters	45	8.63	1.57	30.42	
PFHxS						
C8 Health Project (2005-2006), Ohio or West Virginia	Male firefighters (median employment 12.5 years)	36	4.6	0.25	14.6	Jin *et al*. (2011)
	Other employment	5,373	3.60	0.25	1,447.6	
FOX Study (2010-2011), California	Firefighters	101	2.27	NR	13.2	Dobraca *et al*. (2015)
	NHANES (2009-2010)	876	2.20	NR	44.8	
NHANES (2018-2019), Southwest Ohio	Airport firefighters	36	6.45	2.2	12.28	Leary *et al*. (2019)
	Suburban firefighters	9	3.15	0.84	22.49	
	Combined firefighters	45	6.15	0.84	22.49	
PFNA						
C8 Health Project (2005-2006), Ohio or West Virginia	Male firefighters (median employment 12.5 years)	36	1.6	0.25	4.4	Jin *et al*. (2011)
	Other employment	5,373	1.5	0.25	11.6	
FOX Study (2010-2011), California	Firefighters	101	1.13	NR	4.23	Dobraca *et al*. (2015)
	NHANES (2009-2010)	876	1.31	NR	17.95	
NHANES (2018-2019), Southwest Ohio	Airport firefighters	36	0.45	0.22	1.36	Leary *et al*. (2019)
	Suburban firefighters	9	0.47	0.20	0.57	
	Combined firefighters	45	0.46	0.20	1.36	

Notes:
FOX = Firefighter Occupational Exposures; Min = Minimum value reported; Max = Maximum value reported; N = Number of participants/samples; NHANES = National Health and Nutrition Examination Survey; PFAS = Perfluoroalkyl Substances; PFHxS = Perfluorohexane Sulfonate; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctane Sulfonate; PFNA = Perfluorononanoic Acid.

7 Variability in Factors that Influence Toxicity

In this section I describe variability in factors that can influence the toxicity of different PFAS. I use examples from human studies, when available, as well as relevant examples from animal studies. I discuss variability in the relationship between exposure to different PFAS and serum concentrations among humans. I also discuss how exposures to different PFAS can result in different toxicological responses, relying in particular on data from animal studies, as doses in these studies can be closely controlled and monitored throughout the course of a study, and effects can be more readily identified and quantified than in human studies . Variability among individuals in the factors that influence PFAS doses and responses in the body indicates that the evaluation of the potential health effects of exposure to PFAS (if any) would require assessment of exposure and dose, and hence risk, on an individualized basis. As stated above in Section 5, I understand that considering variability among individuals is part of the class certification process, even though it is my opinion that the overall evidence does not demonstrate that humans have been harmed by PFAS exposure (see, for example, ATSDR, 2018a; Australia, Expert Health Panel for PFAS, 2018; Steenland *et al*., 2020).

7.1 Toxicokinetics

Toxicokinetics is the study of the processes of absorption, distribution, metabolism, and excretion (also referred to as ADME). Collectively, these processes determine the concentration of a chemical in target tissues where health effects occur. ADME can differ both within and across species, and thus, given the same administered dose, differences in ADME can result in differences in target tissue chemical concentrations. In this section, I focus on sources of variability in toxicokinetics among humans, which can result in differences in serum concentrations[8] and in tissue concentrations. Differences in toxicokinetics among different PFAS and among different human populations (*e.g*., pregnant women) provide support for the need for individualized determinations of exposure, dose, and risk among humans.

7.1.1 Absorption

Absorption describes the movement of a substance into the bloodstream, with the speed and extent to which a substance is absorbed depending on several factors, including the particular route of exposure and the physicochemical properties of the substance (Slitt, 2019). Insight as to how and to what extent a substance is absorbed affects other parameters, such as bioavailability and tissue dose, that will ultimately impact potential toxicity and overall body burden.

The majority of available data regarding PFAS absorption is from studies in rats. These studies indicate that PFOA and PFOS are well absorbed following oral exposure, with estimated absorption fractions of > 90% (Chang *et al*., 2008a; ATSDR, 2018a; US EPA, 2016d,e). Absorption data are limited for other PFAS, though PFBA, PFHxS, PFNA, and PFDA have also been shown to be well absorbed orally in animals (Chang *et al*., 2008a; Kim *et al*., 2016, 2018, 2019; Fujii *et al*., 2015). There is also evidence that PFOA is absorbed by rats following both inhalation and dermal exposure, but to a lesser extent than following oral exposure (Kennedy, 1985; Kennedy *et al*., 2004).

[8] I note that there are also important toxicokinetic interspecies differences that support the use of serum concentration, rather than exposure dose, for developing health-based criteria based on animal studies.

As discussed by ATSDR (2018a), observations that PFOA concentrations in serum or plasma are related to PFOA exposure *via* drinking water provide evidence of oral absorption of PFOA in humans (*e.g*., Hoffman *et al*., 2011; Seals *et al*., 2011; Bartell *et al*., 2010; Holzer *et al*., 2008; Wilhelm *et al*., 2008). By contrast, PFOA does not appear to be well absorbed through human skin (Fasano *et al*., 2005). I did not identify any data regarding absorption of PFOA *via* inhalation or of other PFAS *via* any exposure route in humans.

Overall, the limited data regarding absorption indicate that the specific PFAS studied are likely well absorbed after ingestion but are not well absorbed *via* inhalation or dermal contact. Nonetheless the available data, mainly from studies of PFOA and PFOS, indicate that the internal concentration in the body after exposure to these PFAS may differ based on the exposure route.

7.1.2 Distribution

Distribution of a chemical in the body describes the transfer of a chemical throughout the body across different compartments (*e.g*., blood, specific tissues). Distribution processes are typically reversible and ultimately determine the free concentration of the chemical in the blood or tissues (Slitt, 2019). The circulating or tissue concentration dictates the onset and intensity of any potential toxicity observed after exposure. As with other toxicokinetic parameters, a better understanding of distribution patterns may help elucidate observed differences in toxicity across different PFAS, either among or within species, and may also help predict whether such differences might exist in the absence of information to that effect. In this section, where appropriate, I present data from animal studies because of the limited information in humans for some parameters.

As shown in Table 7.1, while there are similarities in overall PFAS tissue distribution, there are also notable differences that appear to vary by specific PFAS, species, and, to a lesser extent, by sex. The overall evidence suggests that PFOA, PFOS, and PFBS preferentially distribute to the liver in several species and do not readily cross the mature blood-brain barrier. In contrast, PFBA and PFHxS appear to preferentially distribute to the serum and, to a lesser extent, to the liver in animals. The data for humans are limited but do not show significant concentrations of PFOA or PFOS in the liver at environmental doses (Olsen *et al*., 2003b 203-3119). Data from Harada *et al*. (2007), in which PFOA and PFOS cerebral spinal fluid concentrations were more than 500-fold lower than serum concentrations in adult humans, provide additional evidence that these compounds do not readily cross the mature blood-brain barrier in humans. It is important to note that data for all species aside from rats, and for the majority of PFAS in general, are limited. Nonetheless, the available data are indicative of differences in tissue distribution among different PFAS.

Table 7.1 Tissue Distribution of PFAS by Species and Sex

Species	Sex	PFBA	PFBS	PFOA	PFOS	PFHxS	PFNA
Rats	Males	Serum >> Liver[a]	N/A	≤ 5 mg/kg Liver >> Serum > Kidney >> Spleen>> Brain[b] ≥ 10 mg/kg Serum ≥ Liver ≥ Kidney >> Spleen >> Brain[b]	≤ 0.3 mg/kg Liver >>> Serum ≈ Kidney >> Spleen >> Brain[c] 2 mg/kg Liver >> Serum >> Kidney >> Spleen[d]	Serum >> Liver > Kidney >> Spleen[e]	Liver >> Serum > Kidney[f]
	Females	N/A	N/A	≤ 5 mg/kg Serum ≈ Liver ≥ Kidney >> Spleen[b] ≥ 10 mg/kg Serum > Kidney > Liver >> Spleen >>> Brain[b]	2 mg/kg Liver >> Serum >> Kidney >> Spleen[d]	Serum >> Liver > Kidney >> Spleen[e]	Serum >> Kidney > Liver[f]
Mice	Males	Serum >> Liver[a]	Liver > Kidney >> Spleen >> Brain[g]	Liver > Serum[h]	Liver >> Kidney >> Spleen >> Brain[i]	Serum >> Liver >> Kidney[e]	Liver > Serum > Kidney[f]
	Females	N/A	N/A	Liver >> Serum[h]	N/A	Serum >> Liver >> Kidney[e]	Liver > Serum > Kidney[f]
Monkeys	Males	N/A	N/A	Serum >> Liver[j]	Liver > Serum[k]	N/A	
Humans	N/A	Kidney >> > Liver >> Brain[l]	Kidney >> Liver (ND in brain)[l]	Liver >> Kidney (ND in brain)[l]	Kidney ≥ Liver >>> Brain[l]	Kidney >> Liver ≥ Brain[l]	Brain ≥ Kidney >> Liver[l]

Notes:
N/A = Not Available; ND = Not Detected; PFAS = Perfluoroalkyl Substances; PFBA = Perfluorobutanoic Acid; PFBS = Perfluorobutane Sulfonate; PFHxS = Perfluorohexane Sulfonate; PFNA = Perfluorononanoic Acid; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctane Sulfonate.
≥ indicates tissue concentration is comparable or slightly greater than tissue concentration in comparison tissue.
> indicates there is a less than 2-fold difference in relative tissue concentration.
>> indicates there is a 2- to 10-fold difference in relative tissue concentration.
>>> indicates there is a greater than 10-fold difference in relative tissue concentration.
(a) Chang *et al*. (2008a).
(b) Data from multiple studies (a more complete description of the values and their sources can be found in Pizzurro *et al*., 2019); data for PFOA levels in female rat brain at low doses are not available.
(c) Iwabuchi *et al*. (2017).
(d) Kim *et al*. (2016).
(e) Benskin *et al*. (2009).
(f) Tatum-Gibbs *et al*. (2011) and Kim *et al*. (2019) for males only.
(g) Bogdanska *et al*. (2014).
(h) Data from one male and one female mouse (Hundley *et al*., 2006).
(i) Bogdanska *et al*. (2011).
(j) Butenhoff *et al*. (2004); evaluated only serum and liver concentrations.
(k) Seacat *et al*. (2002); evaluated only serum and liver concentrations.
(l) Data from 20 human cadavers (Perez *et al*., 2013). Note that this study has significant limitations, including the small number of individuals studied and the lack of removing blood from the tissues prior to measuring tissue PFAS levels.

Studies of humans and laboratory animals demonstrate that at least some PFAS can cross the placenta, referred to as placental transfer, and into breast milk, referred to as lactational transfer. Thus, developing fetuses may be exposed to PFAS *in utero*, and newborns may be exposed to PFAS *via* lactation. As shown in Table 7.2, although placental and lactational transfer of PFOA are comparable in rats and humans, PFOA blood concentrations in offspring relative to maternal blood concentrations are greater in humans than in rats. Similarly, for PFOS, blood concentrations in offspring relative to maternal blood concentrations are somewhat greater in humans compared to rats, despite the fact that placental and lactational transfer of PFOS is considerably greater in rats compared to humans. The basis for this is not clear but could be related to differences in biological half-lives for PFOA and PFOS, which are considerably longer in humans compared to rats (as discussed further below in Section 7.1.4). Table 7.2 also shows that the ratios of blood concentrations in human offspring relative to maternal blood concentrations are variable across PFAS. According to ATSDR (2018a), these ratios depend on the structure of the PFAS, with longer chain length and a terminal sulfonate group generally being associated with lower ratios.

Table 7.2 Placental and Lactational Transfer

PFAS	Placental Transfer[a]		Lactational Transfer[b]		Offspring/Maternal Ratio[c]	
	Humans	Rats	Humans	Rats	Humans	Rats
PFOA	0.79 (0.62-1.5)	0.42	0.04 (0.03-0.12)	0.10	3.6 (2.7-4.6)	0.26
PFOS	0.37 (0.29-0.56)	2.3	0.01 (0.01-0.03)	0.31	0.88 (0.72-1.4)	0.68
PFHxS	0.56 (0.23-1.25)	N/A	0.010 (ND-0.020)	N/A	1.6 (1.2-2.0)	N/A
PFNA	0.50 (ND-1.25)	N/A	0.010 (ND-0.048)	N/A	2.0 (Single Value)	N/A

Notes:
N/A = Not Available; ND = Not Detected in Offspring; PFAS = Perfluoroalkyl Substances; PFHxS = Perfluorohexane Sulfonate; PFNA = Perfluorononanoic Acid; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctane Sulfonate.
A more complete description of the values and their sources can be found in Pizzurro *et al*. (2019). PFNA values are from ATSDR (2018a) and Fromme *et al*. (2010).
(a) Presented as fetal (cord blood)/maternal (serum or plasma) ratio. Values for humans represent median and range. Value for rats selected as lowest dose (as being most comparable to exposure and sample collection in humans).
(b) Presented as breast milk/serum (or plasma) ratio. Values for humans represent median and range. Values for rats selected as lowest dose and at earliest time-point (as being most comparable to exposure and sample collection in humans).
(c) Values for humans represent average of two studies, using earliest postnatal time point, based on PFAS concentrations quantified in serum or plasma. Values for rats selected as lowest dose at earliest postnatal time point; based on PFAS concentrations quantified in either serum or plasma.

Overall, the studies of PFAS distribution indicate that different PFAS can vary in their distribution to different organs and tissues in the body, which can affect their bioavailability and susceptibility to potential effects on specific organs. This variability in distribution, as well as in placental and lactational transfer, can also result in variability in serum concentrations across different PFAS and across species, sexes, doses, and life stages. Accordingly, evaluating risk (if any) in individuals exposed to PFAS requires an exposure/dose assessment for the specific PFAS at issue and cannot be generalized for all PFAS together.

7.1.3 Metabolism

Most PFAS do not appear to undergo metabolism in the liver or other tissues (ATSDR, 2018a; US EPA, 2016d,e). PFAS are rather unusual in this regard, because liver metabolism is common for many chemicals.

Potential detoxification or bioactivation[9] in the liver often complicates the extrapolation of health effects across species, because different species sometimes show differences in metabolism. There is also the potential for differences in metabolism among members of the same species (*e.g.*, ethnic differences in the ability to metabolize ethanol among humans).

Although the presence of a particular PFAS in the serum may be indicative of exposure to that specific PFAS compound, it may also indicate exposure to a precursor PFAS compound. For example, PFOS can be produced from the metabolism of 2-(N-ethyl-perfluorooctane sulfonamido) acetic acid, 2-(N-methyl-perfluorooctane sulfonamido) acetic acid, or PFOSA (Olsen *et al.*, 2005; 3M, 2000). PFOA can be produced from the metabolism of 8-2 fluorotelomer alcohol (Henderson and Smith, 2007; Kudo *et al.*, 2005). PFNA has been shown to be produced from the metabolism of 8-2 telomer alcohol in mice (Kudo *et al.*, 2005). Thus, variability in exposures to these parent compounds, as well as potential differences among humans in the ability to metabolize them, can contribute to variability in serum concentrations of PFOS, PFOA, or PFNA. Identifying the potential source or sources of any serum PFAS concentration in a given individual must take potential metabolism of precursor compounds into account and generally requires an individualized exposure assessment.

7.1.4 Excretion

Elimination rate is characterized by an elimination half-life, which is the time required for half of the total amount of a chemical to be eliminated from the body, assuming there is no ongoing exposure. Approximately 97% of the total amount of a chemical in the body will be eliminated in five half-lives (*i.e.*, $1\text{-}0.5^5$). A related term is clearance, which refers to the rate at which a chemical can be removed from blood plasma (*e.g.*, by metabolism or excretion). Clearance is typically measured in units of liters of blood plasma cleared of the substance per hour (Krishnan, 2019). Because the body has a fixed amount of blood plasma (about 3 L in an adult human), knowing the clearance allows one to estimate how long a chemical will remain in the body.

In humans, both urinary and biliary clearance[10] are important in the excretion of PFAS (Harada *et al.*, 2007; Zhang *et al.*, 2015). Urine is also a primary elimination pathway for PFOA and PFOS in rats (E.I. Du Pont de Nemours and Co., 2003; Chang *et al.*, 2012). Table 7.3 summarizes the elimination half-lives for several PFAS across species and sexes, and indicates shorter half-lives in female rodents and monkeys compared to males for many, but not all, of the PFAS shown. Although the human data for individual sexes are more limited than for animals, human monitoring studies have not consistently detected sex differences in half-lives of PFAS (Bartell *et al.*, 2010; Seals *et al.*, 2011; Chang *et al.*, 2008b; Wong *et al.*, 2014; Li *et al.*, 2018). Thus, excretion rates of PFAS in humans do not explain the lower serum concentrations of PFAS reported in females in the general population compared to males, as discussed in Section 6.1. Other differences related to excretion between males and females that may explain the sex differences in serum concentrations of PFAS are discussed below.

[9] Bioactivation involves enzymatic alteration of a parent compound to a more biologically active and potentially more toxic compound.

[10] Biliary clearance describes the process by which substances are excreted into bile in the liver, removing the substance and its metabolites from the body prior to entering general circulation. Depending on the physicochemical characteristics of the substance, substances excreted into bile may be transported to the intestine and excreted *via* feces or circulate between the liver and the intestines *via* a process referred to as enterohepatic circulation (Slitt, 2019).

Table 7.3 PFAS Elimination Half-Lives[a]

Species	Sex	PFBA	PFBS	PFOA	PFOS	PFHxS	PFNA	PFDA	PFHxA
Humans[b]	Females/ Males	3.1 days	25.8 days	2.1-8.5 years	3.3-5.4 years	5.3-15.5 years	2.5-4.3 years	N/A	N/A
Monkeys	Females	1.7 days	3.5 days[c]	32.6 days	110 to ~200 days	87 days	N/A	N/A	2.4 days (iv exposure)
	Males	1.7 days	4.0 days[c]	~20 days	132 to ~200 days	141 days	N/A	N/A	5.3 days (iv exposure)
Rats	Females	1.0 hours (iv exposure) 1.8 hours (oral exposure)	0.64-7.4 hours	1.9-4.6 hours (< 25 mg/kg) 16.2 hours (25 mg/kg) 24 hours (50 mg/kg)	24-83 days	0.9-2.0 days	32 days (oral exposure)[d] 4.44 days (iv exposure)[i]	1,406 hours (iv exposure) 1,080 hours (i.p. exposure)[e] 1,200 hours (iv exposure)[i]	2.5 hours (iv exposure)[f] 2.6 hours (50 mg/kg) 2.2 hours (150 mg/kg) 2.1 hours (300 mg/kg)
	Males	6.4 hours (iv exposure) 9.2 hours (oral exposure)	2.1-4.7 hours	1.6-15 days (< 25 mg/kg)[g] 6.5 days (25 mg/kg) 4.4 days (50 mg/kg)	26-82 days[h]	16-34 days	24-42 days (oral exposure)[d] 40.2 days (iv exposure)[i]	958 hours (iv exposure) 552 hours (i.p. exposure)[e] 2,626 hours (iv exposure)[i]	2.1 hours (iv exposure)[f] 2.2 hours (50 mg/kg) 2.4 hours (150 mg/kg) 2.5 hours (300 mg/kg)
Mice	Females	2.9 hours (10 mg/kg) 3.1 hours (30 mg/kg) 2.8 hours (100 mg/kg)	N/A	1.2 days (20 mg/kg-day) 15.6 days (1 or 10 mg/kg)	38 days (1 mg/kg-day) 30 days (20 mg/kg-day)	25-27 days	26-69 days[d]	N/A	N/A
	Males	13.3 hours (10 mg/kg) 16.3 hours (30 mg/kg) 5.2 hours (100 mg/kg)	N/A	21.7 days (1 or 10 mg/kg)	43 days (1 mg/kg-day) 36 days (20 mg/kg-day)	28-30 days	34-228 days[d]	N/A	N/A

Notes:
i.p. = Intraperitoneal Injection; iv = Intravenous; N/A = Not Available; PFAS = Perfluoroalkyl Substances; PFBA = Perfluorobutanoic Acid; PFBS = Perfluorobutane Sulfonate; PFDA = Perfluorodecanoic Acid; PFHpA = Perfluoroheptanoic Acid; PFHxA = Perfluorohexanoic Acid; PFHxS = Perfluorohexane Sulfonate; PFNA = Perfluorononanoic Acid; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctane Sulfonate.
A more complete description of the values and their sources can be found in Pizzurro *et al*. (2019).
(a) Unless otherwise specified, data represent both oral and iv exposures, both acute and chronic exposures, and a range of doses.
(b) PFBA data from Chang *et al*. (2008a) represent geometric mean for three employees from the Cottage Grove, Minnesota, facility and nine employees from the Cordova, Illinois, facility. PFBS data from Olsen *et al*. (2009). PFNA data from Zhang *et al*. (2013). PFDA data from Vanden Heuvel *et al*. (1991) and Ohmori *et al*. (2003). PFHxA data from Chengelis *et al*. (2009). PFHpA data from Ohmori *et al*. (2003).
(c) Mean terminal serum elimination half-life ($T_{0.5}\gamma$) for PFBS is from Olsen *et al*. (2009), estimated using a three-compartment model. Note that Chengelis *et al*. (2009) reported elimination half-lives of 8.1 and 15 hours, respectively, for males and females. However, they monitored serum PFBS concentrations for only 7 days *vs*. 31 days as in Olsen *et al*. (2009). Hence, Chengelis *et al*. (2009) might not have captured the third phase of elimination observed by Olsen *et al*. (2009), reflected in the much longer half-lives estimated by Olsen *et al*. (2009).
(d) Data represent a range of single oral doses from 1 to 10 mg (Tatum-Gibbs *et al*., 2011).
(e) Data from Vanden Heuvel *et al*. (1991).
(f) Data from Chengelis *et al*. (2009).
(g) Excludes data from Johnson and Ober (1980). Although the elimination half-life as reported by ATSDR (2018a) was 4.8 days, the basis for estimating the half-life was not clear from the underlying data provided in Johnson and Ober (1980).
(h) Excludes data from Johnson and Ober (1979). Although the elimination half-life as reported by ATSDR (2018a) was 7.5 days, the basis for estimating the half-life was not clear from the underlying data provided in Johnson and Ober (1979).
(i) Data from Kim *et al*. (2019) for PFNA (3 mg/kg) and PFDA (1 mg/kg).

As shown in Table 7.3, half-lives are generally longer for the 6- to 9-carbon PFAS *vs*. the 4-carbon PFAS and are also generally longer for the sulfonates *vs*. the carboxylates. There are substantial differences in PFAS elimination rates between humans, monkeys, and rats, with much longer half-lives found in humans. As discussed by Harada *et al*. (2007), the long half-lives for PFOA and PFOS in humans may be due to low levels of urinary excretion coupled with a high rate of biliary reabsorption. Reabsorption from kidney tubules by organic anion transporter (OAT) 4 and urate transporter 1 may also contribute to the long biological half-life of PFOA in humans (Nakagawa *et al*., 2009; Yang *et al*., 2010).[11] In contrast to humans, in which the ranges of half-lives of PFOA and PFOS are comparable, the half-life of PFOA in monkeys and rats is considerably shorter than the half-life of PFOS.

In addition to urine and feces, menstruation and lactation can be important elimination routes in women. Using a physiologically based pharmacokinetic (PBPK)[12] model, Wong *et al.* (2014) estimated that the serum elimination half-life of PFOS was significantly lower in women (4 years) compared to men (4.7 years) and determined that menstruation explains 30% of this difference. Kang *et al.* (2016) observed an inverse correlation between the PFOA breast milk concentration and length of lactation and suggested that lactation may be an important excretion route for lactating women. Other studies show that serum concentrations of PFOA, PFOS, and PFNA are lower in women who breastfeed compared to women who do not breastfeed, with maternal serum decreasing approximately 2-3% per month of breastfeeding (Brantsaeter *et al*., 2013; Mondal *et al*., 2014). The limited data available for PFHxS do not indicate a comparable effect (Kingsley *et al*., 2018; Mondal *et al*., 2014), and information on the effects of breastfeeding is not available for other PFAS.

PBPK models that evaluate the elimination of PFOA and PFOS through lactation in humans were developed to explore the ways in which physiological changes associated with development affect the pharmacokinetics of these compounds in the mother, fetus, and infant (Verner *et al*., 2016; Loccisano *et al*., 2013). Both models incorporate elements of placental and lactational transfer of PFOA and PFOS to the developing fetus and infant. While the models differed somewhat in their construction and data sources, they both predicted approximately 3-4 times higher PFOA plasma concentrations in breastfeeding infants as compared to the mothers at 6 months post-birth and similar or only slightly increased PFOS plasma concentrations in breastfeeding infants at 6 months or children at 3 years compared to concentrations in lactating mothers at the same time point.

Individuals can vary in their serum concentrations of PFAS based on their underlying health status, as certain conditions may affect the excretion of PFAS from the body. For example, menopausal women who no longer menstruate do not lose PFOA and PFOS from their bodies as quickly as women who are still menstruating, and thus would have relatively higher serum concentrations of these PFAS. In addition, serum PFOA and PFOS concentrations decrease during pregnancy and lactation, despite the lack of menstruation, such that women who are not pregnant or breastfeeding would have higher serum PFOA concentrations than those who are pregnant or breastfeeding. Conditions that affect kidney function may also determine the concentration of PFAS in serum (Verner *et al*., 2015). Low glomerular filtration rate (GFR) is an indicator of reduced kidney function, and lowered kidney function reduces the excretion of PFOA from the body; thus, individuals with lower GFR would have higher serum concentrations of PFOA. Thyroid hormones can influence the GFR, so serum concentrations of PFAS may also vary based on an individual's thyroid hormone levels.

[11] OATs and urate transporter 1 are kidney proteins involved in the excretion of chemicals into the urine and reabsorption of chemicals back into the blood (Weaver *et al*., 2010; Yang *et al*., 2010).

[12] PBPK modeling involves using a computer program to describe and model the ADME of a chemical (or chemicals) in the body (Krishnan and Andersen, 2001).

Overall, there is variability in the elimination rates across different PFAS and across species and sexes, and inter-individual differences in half-lives of specific PFAS across studies of humans. Menstruation and lactation can be important elimination routes in women and may contribute to variability in serum concentrations of PFAS among women depending on whether they menstruate, are pregnant, or are breastfeeding. Underlying conditions related to kidney and thyroid function may also contribute to variability in serum PFAS levels among individuals. These significant sources of variability mean that PFAS doses, which affect the potential risk an individual may have, are likely to differ widely among individuals exposed to the same types and amounts of PFAS and will also vary among specific PFAS for the same levels of exposure in a particular individual. As a result, to be reliably estimated, potential risk must be assessed on an individual basis and for specific PFAS.

7.2 Target Organs

In this section, I discuss target organs from experimental animal studies among different PFAS compounds, with emphasis on the most sensitive endpoints. These endpoints are often described as "critical effects." Because animal studies have been used to develop nearly all health-based criteria for PFAS, I focus on findings from animal studies. Table 7.4 shows a selection of different national and state agencies and the critical endpoints upon which their criteria are based. This table demonstrates that PFAS chemicals do not all share the same target organs in experimental animal models, and that agency criteria for PFAS are based on different target organs and endpoints, both across agencies and across PFAS. For example, some agencies have chosen immunotoxicity as the basis for PFOA and PFOS criteria but not as a basis for any the other PFAS listed in the table. Similarly, thyroid changes are the basis of some agencies' criteria for PFBA, PFBS, and PFHxS but not for the other PFAS. Kidney is listed as the target organ for PFBS by two of the agencies, but none of the other PFAS in Table 7.4 have kidney listed as a target organ. The fact that agencies do not always agree on the target organs and critical endpoints for any given PFAS reflects the uncertainty in what, if any, effects different PFAS have on human health, especially at environmental exposures.

It is also notable that the half-lives identified by the agencies differ across different PFAS. Thus, given the same daily dose, a PFAS with a longer half-life can result in a larger dose over time (typically as reflected in serum concentrations) than a PFAS with a shorter half-life. This variability in half-lives, as well as variability in target organs and endpoints, across different PFAS (as presented in Table 7.4) indicates that all PFAS cannot be grouped together as a single class of chemical to reliably evaluate risk to individuals.[13]

[13] The fact that all PFAS should not be grouped together for evaluating risk is in contrast to the complaint in this case, which proposes that PFOA be combined with all other PFAS for purposes of class determination (Taft Stettinius & Hollister, LLP, 2019b). Such an approach is not supported by an understanding of the toxicity of PFAS compounds, which would require individualized assessments.

Table 7.4 PFAS Critical Endpoints and Half-Lives as Identified by Selected Government Agencies

PFAS	Agency	Critical Effect(s)	Half-life in humans
PFBA	MNDH (2018a)	Liver toxicity; thyroid toxicity, blood toxicity	2.9 days
PFBS	US EPA (2014)	Kidney toxicity	ND
	MassDEP (2018)	Developmental toxicity	26 days
	MI EGLE (2020)	Thyroid hormone changes	27.7 days
	MNDH (2020)	Kidney toxicity	27.7 days
PFHxS	US EPA (2009)	Cholesterol decrease	8.5 years
	ATSDR (2018a)	Thyroid toxicity	8.5 years
	MassDEP (2018)	Body weight decrease, cholesterol decrease, prothrombin time increase	7.3 years
	MI EGLE (2020)	Thyroid hormone changes	5.3 years
	MNDH (2019a)	Thyroid hormone changes	5.3 years
	NHDES (2019)	Reproductive toxicity	4.7 years
PFNA	ATSDR (2018a)	Developmental toxicity	2.5 years
	MassDEP (2018)	Developmental toxicity	3.2 years
	MI EGLE (2020)	Developmental toxicity	3.9 years
	NHDES (2019)	Liver weight	4.3 years
	NJDEP (2018)	Liver weight	ND
PFOA	US EPA (2016d)	Developmental toxicity	2.3 years
	ATSDR (2018a)	Developmental toxicity	3.8 years
	EFSA (2020)	Immunotoxicity	2.3 years
	MassDEP (2018)	Developmental toxicity	3.5 years
	MI EGLE (2020)	Developmental toxicity	2.3 years
	MNDH (2018b)	Developmental toxicity	2.3 years
	NHDES (2019)	Liver weight	2.3 years
	NJDEP (2019a)	Liver weight, testicular tumors	2.3 years
PFOS	US EPA (2016e)	Developmental toxicity	5.4 years
	ATSDR (2018a)	Developmental toxicity	5.4 years
	EFSA (2020)	Immunotoxicity	5.4 years
	MassDEP (2018)	Developmental toxicity	4.8 years
	MI EGLE (2020)	Immunotoxicity	3.4 years
	MNDH (2019b)	Immunotoxicity	3.4 years
	NHDES (2019)	Immunotoxicity	3.4 years
	NJDEP (2019b)	Immunotoxicity	5.4 years

Notes:
ATSDR = Agency for Toxic Substances and Disease Registry; EFSA = European Food Safety Authority; MassDEP = Massachusetts Department of Environmental Protection; MI EGLE = Michigan Department of Environment, Great Lakes, and Energy; MNDH = Minnesota Department of Health; ND = Not Determined; NHDES = New Hampshire Department of Environmental Services; NJDEP = New Jersey Department of Environmental Protection; PFBA = Perfluorobutanoic Acid; PFBS = Perfluorobutane Sulfonate; PFHxS = Perfluorohexane Sulfonate; PFNA = Perfluorononanoic Acid; PFOA = Perfluorooctanoic Acid; PFOS = Perfluorooctane Sulfonate; US EPA = United States Environmental Protection Agency.

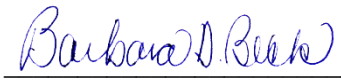
8 Conclusion

There is considerable variability in the factors that influence PFAS exposure, such as the sources, transport, exposure media, and receptors of the various PFAS in the environment. Individual variability factors such as intake, body mass, age, gender, pregnancy status, and (in the case of certain PFAS) menstruation are some of the critical determinants of chemical dose and risk. Intra-individual variability from changes in an individual over time (*e.g.*, physical and behavioral changes), as well as interindividual variability from differences among individuals in a population (*e.g*., age, gender, occupation, diet, race, behavior), will impact PFAS intakes, which will result in variability in dose and risk. There is wide variability in serum concentrations of PFAS across general, occupational, and firefighter populations, and serum concentrations have been shown to vary by gender, pregnancy status, age, and race. This variability provides empirical support for the proposition that PFAS exposures will vary widely among proposed class members.

Toxicokinetic processes influence PFAS doses and responses in the body, and these processes are variable across different PFAS, resulting in different elimination half-lives for different PFAS. These processes are also variable among individuals depending on pregnancy and breastfeeding status, menstruation, or underlying health conditions, resulting in variability in serum concentrations for different PFAS and across sexes, doses, and life stages, independent of the level of intake of a particular PFAS. PFAS chemicals do not all share the same target organs, and health-based criteria for PFAS from national and state agencies are based on different target organs and critical endpoints. The variability in target organs, endpoints, and half-lives across different PFAS indicates that all PFAS should not be grouped together as a single class of chemical for the purpose of evaluating risk to individuals. Overall, my analysis demonstrates that the extent and magnitude of variability across factors that influence PFAS exposure and dose is such that individualized assessments would be required to perform a scientifically reliable assessment of exposure, dose, and risk of PFAS across proposed class members, as well as to trace any observed PFAS blood levels in any such class members to potential sources.

Declaration

I declare under penalty of perjury that the foregoing report is true and correct. Executed on December 11, 2020.



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Appendix A

***Curriculum Vitae* of Dr. Barbara D. Beck**

Barbara D. Beck, Ph.D., DABT, ATS, ERT
Principal
bbeck@gradientcorp.com

Areas of Expertise

Risk assessment, exposure assessment, toxicology, metals, inhaled pollutants, soil contaminants, historical knowledge of toxicology.

Education & Certifications

Ph.D., Molecular Biology and Microbiology, Tufts University, 1976

A.B., Biology, Bryn Mawr College, 1968

Diplomate, American Board of Toxicology (DABT), 1988; recertified 1994, 1999, 2004, 2009, 2014, 2019, current certification valid through 2024

Fellow, Academy of Toxicological Sciences (ATS), 2002; recertified 2007, 2012, 2016

Past President, Academy of Toxicological Sciences, June 2010-June 2011

EU Registered Toxicologist (ERT) *via* membership in the UK Register of Toxicologists, 2004; recertified 2007, 2009, 2012, 2015, 2016, 2017, 2018, 2019, 2020

Professional Experience

1987 – Present GRADIENT, Boston, MA
Principal. Environmental consulting practice includes evaluation of chemical toxicity, health risk assessment for cancer and non-cancer endpoints, review of animal toxicology studies, and multi-media assessment of exposure to environmental chemicals. Special emphasis on metals and inhaled chemicals.

1985 – 2018 HARVARD T.H. CHAN SCHOOL OF PUBLIC HEALTH (formerly Harvard School of Public Health), Boston, MA
Visiting Scientist in the Molecular and Integrative Physiological Sciences Program in the Department of Environmental Health.

1985 – 1987 REGION I, ENVIRONMENTAL PROTECTION AGENCY, Boston, MA
Regional Expert in Toxicology and Supervisory Scientist, Air Toxics Staff. Performed risk assessments for toxic air pollutants. General staff responsibilities included air impacts at waste sites, state air toxic programs, and US EPA radiation programs.

1979 – 1985 HARVARD SCHOOL OF PUBLIC HEALTH (now Harvard T.H. Chan School of Public Health), Boston, MA
Research Associate in Environmental Science and Physiology and Fellow in Interdisciplinary Programs in Health. Developed short-term animal bioassay for pulmonary toxicants. Editor and author of monograph on variations in susceptibility to inhaled pollutants for both cancer and non-cancer endpoints.

1978 – 1979 TUFTS UNIVERSITY SCHOOL OF MEDICINE, Boston, MA
Instructor in Protein Chemistry. Isolated phagocytosis inhibiting factor from immunoglobulin of individuals with inherited susceptibility to bacterial infections.

1977 – 1978 HARVARD UNIVERSITY, Cambridge, MA
Postdoctoral Fellow in Biology. Researched novel properties of bacterial protein elongation factor, EF-Tu, relevant to possible role as a structural protein.

1975 – 1976 UNIVERSITY OF MASSACHUSETTS MEDICAL SCHOOL, Worcester, MA
Postdoctoral Fellow in Microbiology. Isolated and analyzed messenger RNA from slime molds. Initiated project on elongation factor, EF-Tu. Awarded post-doctoral fellowships from the American Cancer Society and the Cystic Fibrosis Foundation.

1968 – 1969 TUFTS UNIVERSITY SCHOOL OF MEDICINE, Boston, MA
Research Assistant in Molecular Biology and Microbiology. Performed genetic and biochemical studies on bacterial lipopolysaccharide.

Professional Activities

- Member, U.S. EPA Science Advisory Board (SAB), 2019 to present, Vice-Chair 2020 to present.
- Member, Recruitment Committee, Academy of Toxicological Sciences, 2017-2019.
- Member, AAAS Mentoring Award Committee, Society of Toxicology Women in Toxicology, 2018.
- Member, Task Force on the Impact of Toxicology on Public Health, Society of Toxicology, 2017.
- Member, Task Force on Low-Level Arsenic Exposure, Society of Toxicology, 2016-2017.
- Member, Candidate Identification Subcommittee of the WIT Nominating Committee, 2016.
- Member, Awards Committee, Society of Toxicology, May 2013-April 2015.
- Member, Massachusetts Department of Public Health Eastern Equine Encephalitis (EEE) Expert Panel, January-May 2012.
- Member, National Research Council's Committee on the Future Options for Management in the Nation's Subsurface Remediation Effort, November 2009-2012.
- President, Academy of Toxicological Sciences, July 2009-June 2010.
- Recipient, Lifetime Achievement Award, Awarded by the University of Massachusetts Amherst, School of Public Health and Health Sciences, 2009.
- Member, Massachusetts Department of Public Health Advisory Committee, 2007.
- Member, Executive Committee, International Dose-Response Society, 2006-April 2013.
- Councilor, Metals Specialty Section, Society of Toxicology, 2006-2007.
- Member, Board of Directors, Academy of Toxicological Sciences, 2005-2011.
- Member, Scientific Advisory Committee to the Manganese Health Research Program, 2004-2009.
- Member, Peer Review Committee, US EPA National Health and Environmental Effects Research Laboratory, Experimental Toxicology Division, 2003.
- Member, CIIT Science Advisory Committee, 2002-2004.
- Member, Program Committee, Society of Toxicology, 2001-2005.
- Member, American Chemistry Council Risk Assessment Methods Technical Implementation Panel, 1998-2001.
- Member, International Life Sciences Institute Steering Committee on Cumulative Risk Assessment, 1998.
- Member, Membership Committee, Society of Toxicology, 1997-2000.
- Member, American Water Works Association Research Foundation Peer Review Panel on Arsenic, 1997-1998.
- Member, Advisory Committee to Public Health Program, Florida A & M University, 1996-2002.
- Member, Risk Assessment Task Force, Society of Toxicology, 1996-2000; Chair, 1999-2000.
- Member, Continuing Education Committee, Society of Toxicology, 1996-1997.
- Member, Watertown, MA, Board of Health, 1995-Present; Former Chair.

- Chair of Session, Ecological and Human Health Protocols at GRI Meeting on Environmentally Acceptable Endpoints in Soil, Arlington, VA, 1995.
- Session Chair, International Conference on Arsenic, San Diego, CA, 1995.
- Rapporteur, US EPA Meeting on Risk Assessment for Chemical Mixtures, Research Triangle Park, NC, 1994.
- Member, Program Committee, Society of Toxicology, 1993-1996.
- Member, Arsenic Task Force, Society for Environmental Geochemistry and Health, 1993-1995.
- President, Risk Assessment Specialty Section, Society of Toxicology, 1994-1995.
- Vice President, Risk Assessment Specialty Section, Society of Toxicology, 1993-1994.
- Member, Work Group on Arsenic, Society for Environmental Geochemistry and Health, 1993.
- President, Northeast Chapter of the Society of Toxicology, 1992-1993.
- Member, Review Committee, US EPA Workshop on the Methodology for Deriving National Ambient Water Quality Criteria for the Protection of Human Health, 1992.
- Consultant to SAB Committee on Hazardous Air Pollutants, 1991.
- Member, Advisory Committee to Harvard Center for Risk Analysis, 1990-1993.
- Member, Committee on Public Communications, Society of Toxicology, 1990-1992.
- Councilor, Inhalation Specialty Section, Society of Toxicology, 1990-1992.
- Member, Advisory Committee to US EPA on Metal Bioavailability, 1990.
- Member, Technical Committee, Council for Health and Environmental Safety of Soils (CHESS), 1988-1990.
- President, Northeast Chapter, Society for Risk Analysis, 1987-1988.
- Member, Peer Review Committee, US EPA Inhalation RfD Document, 1987.
- Member, Maine Science Advisory Panel, 1986-1990.
- Member, US EPA Risk Assessment Forum, 1986-1987.
- Member, Rhode Island Air Toxics Advisory Committee, 1986-1987.
- Member, Massachusetts Visibility/Public Health Index Peer Review Team, 1986.
- Member, Massachusetts Air Toxics Guidelines Review Committee, 1985-1988.
- Member, Air Toxics Committee, Northeast States for Coordinated Air Use Management, 1985-1987.
- Member, American Academy for the Advancement of Science, 1976 to present.

Professional Affiliations

Academy of Toxicological Sciences; American Association for the Advancement of Science; American Thoracic Society; International Society of Exposure Analysis; Society of Environmental Geochemistry and Health; Society for Risk Analysis; New England Chapter of the Society for Risk Analysis; Society of Toxicology; Northeast Chapter of the Society of Toxicology

Projects

Law Firm: Evaluation of human health risks for petroleum constituents, including TPH, at multiple oil and gas sites; identification of data needs to refine risk assessment.

Law Firm: Evaluation of risks from constituents in sediments and surface water at inland waterway. Constituents included organic mercury and other metals, PCBs, and dioxins.

Law Firm: Evaluation of releases to air and soil from a pigment manufacturing facility in the southeast US and subsequent toxicological analysis of multiple constituents, including arsenic and dioxins. Conducted analysis of serum congener patterns to assess sources of dioxins.

Law Firm: Evaluation of potential human health risks for metals, including arsenic, barium, lead, and zinc, at a site used for manufacture of drilling muds.

US Dept. of Justice: Development of sampling plan and risk assessment for spray drift exposure to pesticides.

Law Firm: Evaluation of human health risks from chemical exposures at crude oil production site in South America.

Law Firm: Evaluation of odors associated with releases from a landfill. Performed human health risk assessment for chemicals in air.

Law Firm: Evaluation of potential risks from manganese in air and soil near manufacturing facility as part of regulatory analysis.

Engineering Company: Evaluation of exposure to hexavalent chromium in outdoor air and soil and need for medical monitoring.

Northeast Utility: Provided public communication to residents in the vicinity of a coal fly ash disposal site regarding potential human health risks associated with coal fly ash.

Health Canada: Peer review of exposure components of pilot program Screening Assessment Document for ethylbenzene. Peer review of toxicological analysis of aniline.

Perchlorate Study Group: Comments on scientific validity of US EPA RfD for perchlorate.

Law Firm: Evaluation of state agency human health risk assessment for chemicals, including TCE, in groundwater, conduct of individual-specific human health risk assessment, and identification of other sources of TCE.

Law Firm: Analysis of toxicity and risks of mercaptan compounds in air; evaluation of odor thresholds in comparison with health-based limits for mercaptan compounds.

Automotive Manufacturing Facility: Risk communication regarding PCBs in soil and sediments; provided assistance to individuals in interpreting their blood PCB levels.

Confidential Client: Evaluated toxicity and risks of diacetyl and of butter flavorings.

Law Firm: Evaluation of health significance to nearby residents of releases to air from an oil refinery during upset conditions.

Law Firm: Evaluated risks from perchloroethylene released into indoor air of nearby residents from a Midwestern manufacturing facility. Also evaluated risks from PCBs in soil.

Gas Utility Companies: Analysis of exposures to and toxicological effects of elemental mercury in air.

Law Firm: Exposure and toxicological causation analysis involving multiple health effects claims and potential exposures to dioxins, benzene and pentachlorophenol at former manufacturing site in the Midwest.

Multiple Industrial Clients: Analysis of historical state of knowledge of asbestos exposure, toxicology, and risks.

Law Firm: Provided technical evaluation of agency estimates of NO_x emissions and risks from heavy duty diesel engines; identified uncertainties in choice of assumptions and potential impact on risk.

Law Firm: Evaluation of effects of lead on different health endpoints, including neurocognitive changes and behavioral effects in children.

US EPA Region I: Compilation and review of air toxics monitoring studies in Region I with respect to adequacy in reflecting human exposure and in identifying relevant sources from a risk perspective.

Law Firm: Evaluation of risks associated with CCA-treated wood. Development of exposure studies for CCA-treated wood.

Massachusetts Attorney General: Presentation on the use of risk assessment for the siting of an energy facility.

Law Firm: Provided comments to a state agency regarding the toxicological significance of exposure to PFOA and PFOS *via* drinking water.

Law Firm: Evaluation of potential short-term and long-term human health risks from metals including zinc and organics (including mineral oils) from possible medical exposures.

Environmental Engineering Company: Human health and ecological risk assessment for PAHs, dioxins, and other compounds at a former chemical R&D facility, including development and oversight of sampling.

EPRI: Synthesis report of arsenic research studies. Toxicological analysis of methylmercury and lead; development of research plan.

Consumer Product Manufacturer: Toxicological evaluation of different preservatives and bittering agents for possible use in a consumer product.

US EPA, Office of Research and Development: Development of toxicity data base for inhalation exposure to the Hazardous Air Pollutants listed under the 1990 Clean Air Act Amendments.

Pesticide Registrant: Evaluation of carcinogenic mode-of-action and EU classification of a biocide.

Law Firm: Designed and conducted human volunteer study to evaluate the transfer of metals from smelter residue to hands. Incorporated data from study into risk assessment.

Wood Preservative Science Council: Evaluation of US EPA Stochastic Human Exposure Dose Simulation model.

Law Firm: Evaluated risks of dioxins, furans, and PCBs associated with impoundments and with fish in a southwest US river for a case involving claims of property damage and personal injury.

Law Firm Representing Municipality and Port Authority: Prepared risk assessment for proposed development at former MGP site, evaluating future exposures to construction workers and residents. Developed risk-based remedial targets.

Electronics Manufacturer: Risk communication to plant employees regarding exposures to TCE and DCE in groundwater.

Law Firm: Provided expert testimony regarding development state of toxicological knowledge over time of coal fly ash, as associated with MGP sites.

Consumer Product Manufacturer: Evaluation of toxicity and odor of hydrogen sulfide and risk communication on hydrogen sulfide at community meetings.

Law Firm: Developed conceptual approach for evaluating dosimetry associated with short-term inhalation exposures to emissions from heavy duty diesel engines.

American Red Cross: Review of toxicity of new blood bag plasticizer and assessment of potential risks to blood product recipients.

Engineering Company/Army Corps of Engineers: Evaluation of health significance of metal exposures, especially iron, at historic mine site in New England.

New Mexico Environment Dept.: Risk assessment for metals at copper mining and smelting site.

Chemical Manufacturers Association: Review of US EPA land disposal regulations Phase IV. Review of ozone risk assessment in US EPA ozone staff paper.

Health Effects Institute: Assessment of literature on carcinogenicity of inhaled diesel exhaust particulates, especially using urine mutagenicity. Review of literature on toxicity of carbon monoxide and effects on individuals with angina. Developed database of air pollutants from automobiles.

Multinational Manufacturer: Risk assessment and risk communication for perchloroethylene in drinking water at operating facility in Asia.

American Petroleum Institute: Role of risk assessment in Superfund remedy selection process and associated costs.

American Chemistry Council: Provided comments to EPA on health effects of ozone.

Pesticide Manufacturer: Evaluation of toxicity and environmental migration of organo-arsenicals. Probabilistic margin-of-exposure analysis for inorganic arsenic.

Industrial Client: Participation in advisory panel regarding health effects of inhaled and ingested hexavalent chromium.

Law Firm: Evaluation of possible air exposures and health studies at former phosphorous manufacturer in Florida.

Law Firm: Provided comments to US EPA on their Health Effects Documents for PFOA and PFOS.

American Lung Association of Maine: Technical advice on health effects of criteria and non-criteria air pollutants. Review of regulatory packages.

International Chemical Manufacturer: Evaluation of cancer classification systems and setting of occupational exposure limits in European countries and organizations.

Boston Medical Center: Coordination of study of potential effects of perchlorate in humans.

Zinc Corporation of America: Risk assessment using both environmental and epidemiological data for lead and cadmium in soil at a Superfund site.

Law Firm Representing a Manufacturing Company: Risk assessment for potential exposure to TCE in drinking water, including use of adjustment for mutagenicity.

Major Canadian Mining Company: Evaluation of arsenic exposure at mining/milling site using biological monitoring, risk assessment for arsenic, and communication with the public and regulatory agencies.

Law Firm Representing Utility Company: Prepared report regarding historical knowledge of toxicity of simple and complex cyanides, and of oxide box waste materials at former MGP sites. Prepared risk assessment for site.

New Jersey Dept. of Environmental Protection: Site assessment and risk assessment for specialty chemical manufacturing site in New Jersey involving volatile organic chemicals and DDT.

Law Firm: Assessment of toxicity and risks of MTBE, especially with respect to tap water exposure.

US EPA/Engineering Company: Development of work plan to conduct morbidity or mortality study, using readily available databases, for high ozone levels experienced in summer of 1988.

Law Firm Representing Smelter Owner: Evaluated health protectiveness of state cleanup levels for arsenic, lead, and cadmium in soil in class action case.

Chemical Manufacturer: Development of risk screening process for evaluating potential hazards at international sites as part of property transfer.

Major Consumer Product Manufacturer: Development and application of adult blood lead model to predict blood lead levels from discontinuous exposures to lead released from a consumer product.

Engineering Company: Risk assessment for lead, asbestos, PCBs, and other chemicals in soil and water at former brake lining manufacturing facility.

Law Firm: Risk support at multiple MGP sites, including evaluation of potential risks from VOCs in groundwater and evaluation of potential risks to workers from PAHs in soil.

Battery Manufacturing Company: Development and oversight on sample collection and analysis program for lead exposure, evaluation of existing blood lead and tooth lead data, and application of blood lead model.

Oil & Gas Company: Risk assessment support for several major mining-related Superfund sites in the western US. Evaluation of toxicology, epidemiology, and bioavailability of metals, including lead, arsenic, and cadmium. Development of cleanup levels.

International Lead Zinc Research and Organization: Development of probabilistic blood lead model.

Marine Shale Processors: Risk assessment of lead, other inorganics and organic compounds in aggregate produced by hazardous waste recycling. Evaluation of risks of air emissions during incineration process.

US EPA Region II: Participated in Monte Carlo exposure and risk analysis of PCBs in fish.

Remedial Trust Representing Consortium of PRPs: Evaluation of university and agency research plans involving groundwater modeling and remedial approaches at former manufacturing site. Evaluation of biomonitoring approaches for metals. Evaluation of arsenic risk assessment for site.

Coalition for Clean Air Act Implementation: Evaluation of technical issues, including use of composite scores, in 112(g), trading of hazardous air pollutants. Quantification of uncertainty in the composite source.

Canadian Mining Company: Risk assessment for multiple metals associated with tailings release at mine in Southeast Asia.

Mining/Smelting Company: Evaluation of multipathway risks associated with slag use. Comments on US EPA Hazardous Waste Identification Rule.

Consortium of Massachusetts Utility Companies: Review of toxicological knowledge of chemicals at MGP sites over time for Massachusetts generic rate setting case.

Law Firm: Evaluation of non-cancer risks from alkylphenols in groundwater at a wood tar site, based on structure activity relationships. Evaluation of risks from polycyclic aromatic hydrocarbons.

Pharmaceutical Company: Comment on *Federal Register* notice on delisting of incinerator ash from RCRA regulations. Reviewed applicability of model to dioxin-contaminated ash.

Northeast States for Coordinated Air Use Management: Technical assistance in organizing conference on use of bioassays in evaluating ambient air pollutants and presentation of report on use of short-term pulmonary bioassays in evaluation of toxicity and potential health effects of urban particulates.

Law Firm: Risk assessment for arsenic-contaminated soil. Assessed human health risks *via* inhalation and ingestion and ecological risks to deer populations.

Gas Research Institute: Assistance in preparation of exposure manual for MGP sites.

Law Firm: Regulatory analysis for perchlorate in drinking water well in Massachusetts.

State Agency: Impact analysis for a potential toxicological exposure of a biologic product related to manufacturing.

Publications – *Articles and Book Chapters*

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National Research Council, Division of Earth and Life Sciences, Water Science and Technology Board, Committee on Future Options for Management in the Nation's Subsurface Remediation Effort. 2012. "Alternatives for Managing the Nation's Complex Contaminated Groundwater Sites." National Academies Press, Washington, DC, 339p. (Dr. Beck was a member of the panel that prepared this report under the auspices of the National Research Council.)

Beck, BD. 2009. "Congress should take risk-based approach to lead-content law." *Wall Street J.* 253(88):A14. April 16.

Valberg, PA; Beck, BD. 1993. "Recalculation of the Arsenic Cancer Slope Factor." Report to IRIS Information Submission Desk (US EPA), August 9.

Beck, BD; Goodman, G, Hemphill, CP. 1993. "Summary of Naphthalene Toxicity Information and Derivation of a Naphthalene Oral RfD." Draft report to US EPA Naphthalene RfD Work Group. August 30.

Beck, BD. 1987. "Acute and Chronic Toxicity of Trichloroethylene," "Non-carcinogenic Risk Assessment," "The Role of Peroxisomal Proliferation in Trichloroethylene Hepatotoxicity and Carcinogenicity." Draft NESCAUM (Northeast States for Coordinated Air Use Management) Health Assessment Document.

Beck, BD. 1982. "The use of bioassays to assess the toxicity of particulates; evaluation of bioassays." In *Appendix 2: Toxic Effects of Airborne Particulates*, p119 - 167, Appendix to Report "Analysis of Health Effects Resulting from Population Exposures to Ambient Particulate Matter." Prepared by Harvard University, Energy and Environmental Policy Center, for US Dept. of Energy, Agreement No. DE-AC02-81EV10731.

Beck, BD. 1975. "*Activity of three murein hydrolases during the cell cycle of Escherichia coli. K-12.*" Ph.D. Dissertation, Tufts University.

Invited Lectures/Other Presentations – 1985-Present

12/19 – "Careers in Toxicology." Presented at the course on Principles of Toxicology: Molecular and Translational Toxicology at the Harvard T.H. Chan School of Public Health, Boston, MA, December 4.

11/19 – "Asbestos and Principles of Toxicology: What You Need to Know" Presented at DRI Asbestos Medicine, Boston, MA, November 13.

12/17 – "YPLL: A Comprehensive Quantitative Tool to Evaluate Worker Risk Under Green and Sustainable Remediatino." Poster # P.13. Presented at the Society for Risk Analysis (SRA) Annual Meeting, Arlington, VA, December 10-14.

11/17 – "Careers in Toxicology." Presented at the course on Principles of Toxicology: Molecular and Translational Toxicology at the Harvard T.H. Chan School of Public Health, Boston, MA, November 29.

12/16 – "Inorganic Arsenic: Health Risk Assessment." Presented at the University of Connecticut, School of Pharmacy, Storrs, CT, December 7.

11/16 – "Careers in Toxicology." Presented at the course on Principles of Toxicology: Molecular and Translational Toxicology at the Harvard T.H. Chan School of Public Health, Boston, MA, November 30.

11/14 – "Careers in Toxicology." Presented at the course on Principles of Toxicology: Molecular and Translational Toxicology at the Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA, November 24.

12/13 – "Inorganic Arsenic: Health Risk Assessment." Presented at the University of Connecticut, School of Pharmacy, Storrs, CT, December 11.

4/13 – "Risk Assessment Approaches and Application of IRIS Values: Consideration of Susceptibility and Variability." Presented at the National Research Council Inorganic Arsenic: Scientific Considerations for Hazard Identification and Dose-Response Analysis Workshop, Washington, DC, April 4.

4/12 – "Dose-Response Assessment for Arsenic: A Case Study for Why the LNT Doesn't Work." Presented at the 2012 Annual Meeting of the International Dose-Response Society, University of Massachusetts, Amherst, MA, April 25.

12/11 – "Inorganic Arsenic: Health Risk Assessment." Presented at the University of Connecticut, School of Pharmacy, Storrs, CT.

4/11 – Testimony of Barbara D. Beck, Ph.D., DABT, Fellow ATS, ERT regarding "Discussion Draft of H.R., a Bill that would Revise the Consumer Product Safety Improvement Act." Submitted to US House of Representatives, Committee on Energy and Commerce, Subcommittee on Commerce, Manufacturing, and Trade Hearing, April 7.

4/10 – "Medical Monitoring and Environmental Exposures." Presented in a session entitled "Presenting Effective Arguments to Courts Against Awarding Medical Monitoring Damages" at the American Conference Institute's Chemical Products Liability and Environmental Litigation Conference, Chicago, IL, April 28.

3/10 – "These Kids are Driving Me Crazy: The Science of Sensitive Subpopulations." Presented at the DRI Toxic Torts and Environmental Law Seminar, New Orleans, LA, March 18.

3/10 – "Weighing Complex Data in Risk Decisions: Concepts of Evidence-Based Toxicology." Roundtable discussion presented at the Society of Toxicology Annual Meeting, Salt Lake City, UT, March 9.

2/10 – "Buckyballs and Nanotubes: Insurance Issues Related to Nanotechnology." Panelist for talk presented at the American Bar Association's 18th Annual Insurance Coverage Litigation Committee Midyear Program. Phoenix, AZ, February 26.

1/10 – "Overview of Arsenic Epidemiology." Presented as part of the EPRI Arsenic Project Scientific Review Panel Meeting, Research Triangle Park, NC, January 28.

12/09 – "Current Risk from Exposure to Nanoparticles." Presented as part of webinar held by Day Pitney, LLP, December 9.

4/09 – "Testimony of Barbara D. Beck, Ph.D., DABT Regarding the Consumer Product Safety Improvement Act: The Need for a Risk-Based Approach." CPSIA Congressional Briefing, Washington, DC, April 1.

3/09 – "What is an Adverse Effect in the Age of 'Omics?" Roundtable discussion presented at the Society of Toxicology Annual Meeting, Baltimore, MD, March 18.

12/08 – "Inorganic Arsenic: Health Risks and Regulations." Presented at the University of Connecticut, School of Pharmacy, Storrs, CT.

5/08 – "Risk Assessment for Essential Metals: Considerations for 'The Path Forward.'" Presented at the Workshop on Health Risk Assessment of Essential Metals, Ottawa, Canada.

5/08 – "Nanotechnology: An Overview of Environmental Health & Safety Issues." Presented as part of webinar held by Day Pitney, LLP.

3/08 – "Challenge in Using Hazard-based Binning in Risk Assessment & Risk Management." Presented at the Society of Toxicology 47th Annual Meeting, Seattle, WA.

1/08 – "Determining Risks to Background Arsenic Using a Margin-of-Exposure Approach." Presented at the Society of Risk Analysis, New England Chapter Meeting, Boston, MA.

10/07 – "An Update on Risk Management Approaches for Nanotechnology: Recent Regulatory Activities and Trends Affecting Product Development." Presented at the Lux Executive Summit, Cambridge, MA.

9/07 – "Is Smaller Always Worse: What Do We Know Now About the Toxicity and Potential Risks of Nanoparticles? What More Do We Need to Know?" Presented at the PANWAT SOT Meeting, Seattle, WA.

7/07 – "The Role of Regulatory Science in Tort Litigation." Presented as part of the Environmental Law Institute's *Critical Developments in Toxic Torts Seminar Series*, Washington, DC.

3/07 – "Using Modeling to Inform the Risk Assessment Process for Arsenic." Presentation at the Society of Toxicology 46th Annual Meeting, Charlotte, NC.

2/07 – "Health Risks of Inorganic Arsenic: A Serious Threat or are there Better Ways to Spend our Resources?" Presented at the Risk Assessment Forum, Yale University, New Haven, CT.

12/06 – "Risk Assessment: An Overview." Presented at the University of Connecticut, School of Pharmacy, Storrs, CT.

12/06 – "Industry Challenges – Getting the Lead Out: Health Risk Considerations." Presented at Plumbing Manufacturers Association and Copper Development Association Roundtable, Chicago, IL.

10/06 – "Use of Nanoscale Zero-Valent Iron (NZVI) Particles for Groundwater Remediation: A Qualitative Risk Assessment." Presented at the University of Massachusetts Soils, Sediments, and Water Conference, Amherst, MA.

9/06 – "Overview of Science Issues Associated with Assessing Lead Health Effects." Presented at the Battery Council International Convention, Tucson, AZ.

4/06 – "Interpretation of Biomonitoring Studies to Assess Exposure and Risk of Inorganic Arsenic: Confounding by Other Sources of Arsenic." Presented at Toxicology and Risk Assessment Conference, Cincinnati, OH.

1/06 – "A Qualitative Risk Assessment to Evaluate the Remediation of Trichloroethylene by Nanoscale Zero-Valent Iron Particles." Presented at the 1st International Conference on Nanotoxicology: Biomedical Aspects, Miami, FL.

12/05 – "Regulation of Nanotechnology in the Environment and Workplace: Comparative Approaches." Presented at the 2005 Materials Research Society Meeting Session S9: Regulation of Nanotechnology and Nanomaterials, Boston, MA.

9/05 – "Recommendations for DMA Assessment." Presented on behalf of MAA Research Task Force, US EPA, Washington, DC.

9/05 – "Characterization of DMA Risk Using a Nonlinear Dose-Response Approach." Presented on behalf of MAA Research Task Force, US EPA, Washington, DC.

3/05 – "The Life of a Consultant." Society of Toxicology 44th Annual Meeting, New Orleans, LA.

3/04 – "Arsenic Methylation: Considerations for Risk Assessment." Society of Toxicology 43rd Annual Meeting, Baltimore, MD.

1/04 – "Lack of Relevance of DMA-Induced Rat Bladder Tumors for Human Risk Assessment: Metabolism and Disposition Studies of DMA and MMA." Presented to Office of Pesticide Programs, US EPA, Washington, DC.

12/03 – "Selected Comments on Draft EPA Exposure & Risk Assessments for CCA-Treated Wood Using SHEDS-Wood Model." Presented at FIFRA SAP Meeting, Washington, DC.

11/03 – "Risk Assessment: An Overview." University of Connecticut, School of Pharmacy, Storrs, CT.

10/03 – "Comparison of a Probabilistic/Mechanistic Approach to a Deterministic/Empirical Approach for Evaluating CCA-Treated Wood Exposures." Presented at 19th Annual International Conference on Soils, Sediments and Water, Amherst, MA.

4/03 – "Evaluation of Potential Human Health Risks from Copper Azole-Treated Wood." Presented at 99th Annual Meeting of the American Wood-Preservers' Association, Boston, MA.

11/02 – "A Case Study of Arsenic Risk Assessment and Risk Management." Presented at NIEHS DERT Science Retreat, Wilmington, NC.

10/02 – "CCA-Treated Wood: Science and Politics." Presented at University of Massachusetts, Amherst.

10/02 – "Research Activities to Refine Human Health Risk Assessment for CCA-Treated Wood." Presented to CPSC, Washington, DC.

8/02 – "Comments on EPA Background Documents Regarding SHEDS-Wood Model." Presented at Science Advisory Panel meeting, Washington, DC.

1/02 – "Principles of Toxicology." Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA.

12/01 – "Risk Assessment: An Overview." University of Connecticut, School of Pharmacy, Storrs, CT.

10/01 – "Comments on EPA Background Documents Regarding CCA-Treated Wood." Presented to Scientific Advisory Panel, Washington, DC.

10/01 – "Statement by Barbara D. Beck re: Arsenic in Drinking Water: An Update on the Science, Benefits and Cost." Presented at Congressional Hearing, Washington, DC.

8/01 – "Focused Evaluation of Health Risks from Exposure to Arsenic Associated with CCA-Treated Wood." Presented to Consumer Product Safety Commission, Washington, DC.

6/01 – "Adult: Child Differences in the Intra-Species Uncertainty Factor: A Case Study Using Lead." Presented at the Fifth Annual Workshop on Evaluation of Default Safety Factors in Health Risk Assessment, UMDNJ-New Jersey Medical School, Newark, NJ.

3/01 – "Risk Assessment for Metals: Physiologically-Based Pharmacokinetic Models for Metals." Society of Toxicology 40th Annual Meeting, San Francisco, CA.

6/00 – "EPRI-Sponsored Arsenic Research Program – Application to Arsenic Cancer Risk Assessment." SEGH Fourth International Conference on Arsenic Exposure and Health Effects, San Diego, CA.

5/00 – Invited Participant/Speaker to the Fourth Annual Workshop on Evaluation of Uncertainty/Safety Factors in Health Risk Assessment, Nutley, NJ.

4/00 – "Development of a Stochastic Physiologically-Based Pharmacokinetic Model for Lead." Toxicology and Risk Assessment Approaches for the 21st Century Conference, Kings Island, OH.

6/99 – "The Development of a Stochastic Physiologically-Based Pharmacokinetic Model for Lead." US EPA Workshop on Lead Model Development: Probabilistic Risk Assessment and Biokinetic Modeling, Raleigh-Durham, NC.

6/99 – "The Development of a Stochastic Physiologically-Based Pharmacokinetic Model for Lead." WHO/IARC Conference on Lead Exposure, Reproductive Toxicity and Carcinogenicity, Gargnano, Italy.

3/99 – "Strategies for Prosecuting and Defending Toxic Tort Litigation – A Toxicologist's Perspective." ABA Annual Conference on Environmental Law, Keystone, CO.

3/99 – "Principles of Toxicology." Harvard Center for Risk Analysis Course "Analyzing Risk: Assessment and Management," Boston, MA.

2/99 – "Comments on EPA Perchlorate RfD Draft Document." Perchlorate Peer Review Workshop, San Bernardino City Council Chambers, San Bernardino, CA.

1/99 – "Risk Assessment: An Overview." Harvard School of Public Health (now Harvard T.H. Chan School of Public Health) Principles of Toxicology Course, Boston, MA.

12/98 – "Risk Assessment: An Overview." University of Connecticut Advanced Toxicology Course, Storrs, CT.

11/98 – "EPA's Proposed Residential Lead Standards." US EPA's Children's Health Protection Advisory Committee Meeting, New Carrolton, MD.

10/98 – "What are the Characteristics of a Well-designed Environmental Lead/Blood Lead Study?" National Environmental Policy Institute's Conference "Protecting Children's Health: Assessing the Relationship of Soil Lead to Blood Lead," Washington, DC.

10/98 – "Principles of Toxicology." Harvard Center for Risk Analysis Course "Analyzing Risk: Assessment and Management," Boston, MA.

10/98 – "Cumulative Risk – Case Study." American Law Institute-American Bar Association Course of Study, "Risk Assessment and Risk Management in Environmental Law," Washington, DC.

10/98 – "Introduction to Recent Developments in Risk Assessment: Aggregate Exposure and Cumulative Risk." American Law Institute-American Bar Association Course of Study, "Risk Assessment and Risk Management in Environmental Law," Washington, DC.

4/98 – "Principles of Toxicology." Harvard Center for Risk Analysis Course "Analyzing Risk: Assessment and Management," Boston, MA.

3/98 – "Impact of Arsenic (As_i) Metabolism on Human Populations: Dose-Response Relationships in Arsenic-Induced Cancers." Society of Toxicology 37th Annual Meeting, Seattle, WA.

3/98 – "Effective Risk Communication: Avoiding the Pitfalls." Continuing Education Course, Society of Toxicology 37th Annual Meeting, Seattle, WA.

12/97 – "Key Issues Raised by EPA's Proposed Ozone Standards and Supporting Analysis." Society for Risk Analysis, Annual Meeting and Exposition, Washington, DC.

5/97 – "Testimony on Analysis of Risk Assessment Used by the EPA in Support of Its Proposed Ozone Standards." Before the Joint Hearing of the Health and Environment Subcommittee and the Oversight and Investigations Subcommittee, Commerce Committee, US House of Representatives, Washington, DC.

3/97 – "Principles of Toxicology." Harvard Center for Risk Analysis Course "Analyzing Risk: Assessment and Management," Boston, MA.

12/96 – "Risk Assessment: An Overview." University of Connecticut Course "Advanced Toxicology", Storrs, CT.

12/96 – "Risk Assessment for Criteria Pollutants *versus* Other Noncarcinogens: The Difference Between Implicit and Explicit Conservativism." Rutgers University 2nd Annual Workshop "The Evaluation of EPA 10X Safety Factors in Health Risk Assessment," Nutley, NJ.

9/96 – "The Quantitative Use of Information on Susceptibility in Risk Assessment: Where is it Working or Not Working? How Can We Make It Better?" Third Annual NHEERL Symposium on Susceptibility and Risk Assessment, Raleigh, NC.

9/96 – "Principles of Toxicology." Harvard Center for Risk Analysis Course "Analyzing Risk: Assessment and Management," Boston, MA.

8/96 – "The Role of Risk Assessments in Superfund." American Bar Association, Orlando, FL.

12/95 – "Use of Monte Carlo Arsenic (As) Model to Predict Distributions of Urine Arsenic at a Mining and Milling Site." Society for Risk Analysis, Honolulu, HI.

10/95 – "Use of Information on Variations in Susceptibility – Ozone." ILSI Risk Science Institute Workshop on Human Variability, Washington, DC.

10/95 – "Evaluation of Health Effects Resulting from Accidental Exposures." Michigan Society for Risk Analysis, Dearborn, MI.

9/95 – "Principles of Toxicology." Harvard Center for Risk Analysis Course on "Analyzing Risk: Assessment and Management," Boston, MA.

6/95 – "Validation of an Arsenic Exposure Model at a Mining and Milling Site through Urinalysis." "Use of an Arsenic Exposure Model at a Gold Mining and Milling Site." Second International Conference on Arsenic Exposure and Health Effects, San Diego, CA.

7/94 – "A Review of Scientific Issues Pertaining to Arsenic." Society for Environmental Geochemistry and Health Conference on Lead and Arsenic Exposure in the Rocky Mountains, Salt Lake City, UT.

5/94 – "Use of Lead Exposure Assessment in the Regulatory Process." International Lead Zinc Research Organization Lead Exposure Assessment Workshop, Research Triangle Park, NC.

3/94 – "Non-linearities in Arsenic Risk Assessment." Boston Risk Assessment Group, Cambridge, MA.

3/93 – "Basic Risk Assessment: Current Developments." Continuing Education Course, Society of Toxicology, New Orleans, LA.

3/92 – "A Review of the Bioavailability of Petroleum Constituents." West Coast Soils and Groundwater Conference, Long Beach, CA.

3/92 – "Bioavailability of Metals and Organics." Workshop on Human Health and Ecological Risk Assessments for Contaminated Sites, Toronto, Canada.

2/92 – "Improvements in Quantitative Noncancer Risk Assessment." Chair of Symposium of Society of Toxicology Meeting, Seattle, WA.

2/92 – "Perspectives on the Development of Soil Cleanup Levels at Mining Sites." Colorado Bar Association, Denver, CO.

11/91 – "Environmental Law Update: Toxic Torts and How Clean is Clean?" Squire, Sanders & Dempsey, Cincinnati, OH.

10/91 – "Risk Assessment for Indoor Air: Evaluating Risks to Susceptible Populations." NATO/CCMS-COST 613 Joint Workshop, Kloster Banz, Bavaria, Germany.

2/91 – "An Update on Exposure and Risks of Lead." Chair of Symposium at Society of Toxicology Meeting.

2/90 – "Inhalation Risk Assessment." Chair of Symposium at Society of Toxicology Meeting.

1/90 – "The Use of Structure Activity Relationships for Alkyl Phenol Risk Assessment." New England Society for Risk Analysis, Boston, MA; RJR/Nabisco, Winston-Salem, NC.

1/90 – "The Use of Structure Activity Relationships in Risk Assessment." Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA; Northeastern University, Boston, MA.

11/89 – "How Protection Levels are Developed and What They Mean." Course on Risk Assessment and Epidemiology for Lawyers, Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA.

9/89 – "An Environmental Health Case Study." Tufts Medical School, Boston, MA.

3/89 – "Impact of Lead Derived from Mining Sources on Blood Lead." Boston Risk Assessment Group, Boston, MA.

2/89 – "Ecological and Health Risk Assessment for Arsenic in Soil." Society of Toxicology, Atlanta, GA.

12/88, 1/89, 9/89, 10/89, and 4/90 – "Risk Assessment for Hazardous Waste Sites, Including a Perspective on Toxic Torts." Executive Enterprises, Inc., Washington, DC, Chicago, IL, Philadelphia, PA, and Orlando, FL.

10/88 – "Ozone Toxicology and Risk Assessment." Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA.

10/88 – "Risk Assessment for Arsenic in Soil." University of Massachusetts, Amherst, MA.

9/88 – "The Use of Animal Bioassays to Assess Lun31 Toxicity." NESCAUM, Princeton, NJ.

6/88 – "Review of Epidemiological and Toxicological Studies on Mining Derived Lead." US EPA, Philadelphia, PA.

3/88 – "Assessment of Impact on Blood Lead of Lead from Mining Sources." US EPA, Washington, DC.

2/88 – "Regulatory Toxicology." Tufts University School of Medicine, Boston, MA.

12/87 – "Risk Assessment for Soil." Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA.

12/87 – "Health Effects of Ozone and the Clean Air Act." New England Chapter for Society for Risk Analysis, Cambridge, MA.

11/87 – "Health Effects of Ozone." Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA.

9/87 – "Health Risk Assessment for Soil." University of Massachusetts, Amherst, MA.

4/87 – "Key Issues in Addressing Adverse Effects of Ozone." University of Massachusetts, Amherst, MA.

1/87 – "Risk Assessment for Dioxin in Soil." MIT, Cambridge, MA.

10/86 – "Pulmonary Toxicology." Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA.

10/86 – "Risk Assessment." University of Massachusetts, Amherst, MA.

10/86 – "Regulatory Toxicology." Tufts University School of Medicine, Boston, MA.

7/86 – "Health Effects of Indoor Air Pollutants." Region I US EPA, Lexington, MA.

6/86 – "Health Effects of Radon." Society of Women Engineers, Hartford, CT.

6/86 – "Contacting the Health and the Medical Community About the Adverse Effects of Ozone." US EPA, Washington, DC.

6/86 – "Animal Toxicology." US EPA Region I, Boston, MA.

4/86 – "Health Effects of Ozone." NESCAUM meeting, Newport, RI.

2/86 – "Pulmonary Toxicology." US EPA Region I, Boston, MA.

12/85 – "Toxicology of Dioxin." US EPA Dioxin Workshop, Lexington, MA.

11/85 – "Animal Bioassays." Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA.

11/85 – "Pulmonary Toxicology." Harvard School of Public Health (now Harvard T.H. Chan School of Public Health), Boston, MA.

10/85 – "Indoor Air Pollution." Air Pollution Control Association Meeting, Enfield, CT.

1/85 – "Indoor Air Pollution." NESCAUM Workshop, Northampton, MA.

Editor

2010 – Present. Associate Editor. *Toxicology and Applied Pharmacology*.

2006 – 2010. Specialty Editor. *Toxicology and Applied Pharmacology*.

1995 – Present. Editorial Board Member. *Human and Experimental Toxicology*.

Beck, BD. 1992. *Trace Substances in Environmental Health - XXV, Proceedings of a Conference Held in Columbia, Missouri, USA, 20-23 May, 1991 [25th Annual Conference]*. Science Reviews, Ltd., Northwood, England. Prepared for University of Missouri, Environmental Trace Substances Research Center; Society for Environmental Geochemistry and Health; US EPA; Southern California Edison, 311p. Supplement to Vol. 14 (1992) of*Environmental Geochemistry and Health*.

Beck, BD. 1987. "Overview: Assessing Health Risks from Contaminated Soils." *Comments Toxicol.* 1(3-4):171-175.

Reviewer

Annals of Internal Medicine; Cancer Research; Environmental Health Perspectives; Environmental Research; Epidemiology; Fundamental and Applied Toxicology; Human and Ecological Risk Assessment; Human and Experimental Toxicology; Journal of Society of Environmental Geochemistry and Health; Regulatory Toxicology and Pharmacology; Toxicological Sciences

Continuing Education Courses

- Mechanistic and Pharmacokinetic Consideration to Aid in Determining Causality: Frontier for Data Integration, Risk Assessment Specialty Section (RASS) webinar, 2020.
- Advances in CRISPR-Cas9 Tools and Applications for Toxicologists, Society of Toxicology, 2020.
- The Male Reproductive Tract: Development, Toxicology, and Pathology, Society of Toxicology, 2020.
- Immunology Basics Webinar Series, University of Connecticut, 2019.
- FASEB & AAMC Presents: Animal Research: Still Necessary for Understanding Human Disease, 2019.
- Developmental Toxicity of the Skeletal System: Interpretation of Findings in DART Studies and Implications for Risk Assessment, Society of Toxicology, 2019.
- Microbiome and Environmental Toxicants: From Study Design and Analysis to Regulatory Guidance, Society of Toxicology, 2019.
- Developmental and Reproductive Toxicity (DART) and Risk Assessment of Environmental Chemicals: Applications, Complexities, and Novel Approaches, Society of Toxicology, 2017.
- Embryology and Developmental Toxicity Testing, Society of Toxicology, 2016.
- Toxicogenomics Meets Regulatory Decision-making: How to Get Past Heat Maps, Network/Pathway Diagrams, and "Favorite" Genes, Society of Toxicology, 2015.
- Current Trends in Genetic Toxicology Testing, Society of Toxicology, 2014.
- Understanding Toxic Neuropathy in Drug Development: Both Clinical and Nonclinical Perspectives, Society of Toxicology, 2013.
- Cutaneous Toxicity: *In Vitro* Methods for Toxicity and Safety Evaluation, Society of Toxicology, 2012.
- Epigenetics in Toxicology: Introduction, Mechanistic Understanding, and Applications in Safety Assessment, Society of Toxicology, 2011.
- Technologies and Tools for Toxicity Testing in the 21st Century, Society of Toxicology, 2010.
- Nanotoxicology: The Science of Developing a Safe Technology, Society of Toxicology, 2008.
- Dose-Response Modeling for Occupational and Environmental Risk Assessment, Society of Toxicology, 2008.
- Toxicology and Molecular Biology of Tissue Repair, Society of Toxicology, 2007.
- Use of Genome Databases for Toxicology, Society of Toxicology, 2006.
- Neuropathology for the Toxicologist, Society of Toxicology, 2006.
- Fundamentals of Nanotechnology: Chemistry, Exposure, Environmental/Health Assessments and Societal Impacts, Society of Toxicology, 2005.
- Integrating Toxicologic Pathology into Compound Evaluation and Risk Assessment, Society of Toxicology, 2002.
- Rodent Toxicity and Nongenotoxic Carcinogenesis: Knowledge-Based Human Risk Assessment from Molecular Mechanisms, Society of Toxicology, 2000.
- An Overview of the Tier 1 Screening Battery Proposed by EDSTAC, Society of Toxicology, 1999.
- Benchmark Dose, Society of Toxicology, 1997.
- Principles of Metal Toxicology, Society of Toxicology, 1997.
- Epidemiology for Toxicologists, Society of Toxicology, 1996.
- International Harmonization: Update on Scientific and Regulatory Issues. Part II: Toxic Substances and Environmental Issues, Society of Toxicology, 1994.
- Case Studies in Risk Assessment: Emphasis on Exposure, Society of Toxicology, 1992.
- Environmental Toxicology, Society of Toxicology, 1991.
- Target Organ Toxicity: Advanced Hepatotoxicity, Society of Toxicology, 1990.
- Toxicity of Agents: Pesticides, Society of Toxicology, 1990.
- Neurotoxicology, Society of Toxicology, 1989.
- Respiratory Tract Toxicology by Classes of Agents, Society of Toxicology, 1988.
- Mid-America Course in Toxicology, 1988.
- Pulmonary Pathophysiology, University of Vermont Medical School, 1979.

Appendix B

Testimony of Dr. Barbara D. Beck, Last 4 Years

Expert Testimony
Barbara D. Beck, Ph.D., DABT, ATS, AAAS Fellow
Through December 2020

Plaintiff (P)	Defendant (D)	Case #	Court	District	Date	Legal Proceeding	Protected?
Ester Warren, *et al.*	Allegheny Ludlum Corp., *et al.*	CV-2003-142.02	Circuit Court	Fayette County, Alabama	12/13/16	Deposition	Unknown
Albin Family Revocable Living Trust, *et al.*	Halliburton Energy Services, Inc.	CIV-16-910-M	District Court	Western District of Oklahoma	8/8/17	Deposition	No
State of Minnesota, by its Attorney General, Lori Swanson, *et al*.	3M Company	27-CV -10-28862	District Court	State of Minnesota, County of Hennepin	11/17/17	Deposition	Yes
Mamdouh Habib and Loris Attia	Adco Cleaning Products, LLC, *et al*.	Case No. BC646594	Superior Court	California Superior Court, Los Angeles County, Central District	6/28/19	Deposition	No
Juan Duarte, *et al*.	United States Metals Refining Company, *et al*.	2:17-CV-O1624-ES-SCM	Superior Court	State of New Jersey, Middlesex County	8/15/19	Deposition	Unknown
Michelle Zangari and Ted Zangari	G&I Energy LLC, *et al*.	MRS-L-000936-17	Superior Court	State of New Jersey, Morris County	10/17/19	Deposition	Unknown
A.O.A., *et al*.	Doe Run Resources Corporation, *et al*.	4:11-cv-00044-CDP	District Court	Eastern District of Missouri, Eastern Division	12/19/19	Deposition	Unknown
Tommy Lindsey, *et al*.	3M Company, *et al*.	5:15-cv-01750-AKK	District Court	North Eastern Division, Northern District of Alabama	1/31/20	Deposition	Unknown
Kowall, *et al*.	United States Steel Corporation, Inc.	2017-3355	District Court	State of Pennsylvania, Washington County	2/4/20	Deposition	Unknown
Kenneth A. Biek and Linda M. Biek	Arconic Inc., et al. United States Steel Corporation	19 L 4656 2020L002893	Circuit Court	Cook County, Illinois	9/30/20	Deposition	Unknown
Kowall, *et al*.	United States Steel Corporation, Inc.	2017-3355	District Court	State of Pennsylvania, Washington Count	11/20/20	Hearing	Unknown
Michelle Baker, *et al.*	Saint-Gobain Performance Plastics Corp., *et al*	Civ. No. 1:16-CV-917	District Court	US District Court, Northern District of New York	12/4/20	Deposition	Unknown